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CONGENITAL DEXTROCARDIA WITH PATENT INTERVENTRICULAR SEPTUM

WITH FLUCROSCOPIC AND ELECTROCARDIOGRAPHIC EXAMINATION *

RUDOLPH DURYEA MOFFETT M.D., AND SELIAN NEUHOF, M.D.
NEW YORK

Congenital dextrocardia is an exceedingly rare condition (Foggie, Graanboom and Osler), one of us (Moffett) being able to find only 126 cases reported in a review of the literature from the year 1649. The exact cause for this malposition does not seem to be clear, though numerous investigators (Pegroux, Kussmaul, Winslow and Maschkox, Seres and Sabatier, Selon Dareste, Fol and Waryuski, von Baer, Bischoff and others) have suggested various theories for this abnormality. The most prominent theory (suggested by Fol and Waryuski) is that in the prenatal development of the heart, the right side develops more rapidly than the left and pulls the heart to that side. No theory is sufficiently convincing to explain all cases. Cases of complete transposition of all the organs are numerous, but dextrocardia alone, as in our case, is exceedingly rare. Almost all of the cases of dextrocardia alone that have been reported present symptoms of congenital defects of the heart itself. This has been corroborated by frequent necropsy reports (Ziegler, Fussell, Probyn-Williams, Ewald, Theremin, Keith, Hochsinger and others). It is interesting to note that most of the cases that have been observed are in the male sex—our case was that of a male child. The early cases of pure dextrocardia were merely clinical reports, but about the middle of the nineteenth century the first postmortem records appear. Schroetter[24] reported a case in 1870 and gave a complete review of the subject.

CASE REPORT

The subject of this paper, D. L., a boy (History No. 3861, 1914, History No. 3009, 1915), came under our observation on Oct. 17, 1914, at the German Hospital Dispensary, New York. The family history is as follows: Number of children living, 7; number of deaths or miscarriages, 3; no history of tuberculosis, rheumatism or syphilis.

* Submitted for publication April 28, 1915.

The previous history of the patient is the following: He was the ninth child, born on the ninth month, the birth being normal; the birth-weight was not recorded. He had had measles and whooping cough during his second year; his appetite was always good; the bowels were regular; no vomiting occurred. He had been breast-fed until the fourteenth month, and then was put on solid food. The mother said that the child had always been "blue" and "short of breath," but never sufficiently dyspneic to remain in bed. When the child was first seen (Oct. 17, 1914) he was 3½ years old and had been brought to the dispensary because of a persistent cough which had lasted several months; the cough was worse during the day while the child was up and about. There were no other symptoms that the mother recognized. Physical examination showed a well-nourished child of 3½ years, with blue lips, active movements of the alae nasi and breathing with its mouth open. The eyes and teeth were normal, but there was blueness and congestion of the blood vessels of the conjunctiva. The hands, feet and lips were distinctly cyanotic; there was distention of the superficial veins of the chest; no thyroid or thymus enlargement; fingers and toes were clubbed (Fig. 1). Weight, 22½ pounds; length of body, 32 inches; circumference of head, 18½ inches; of chest, 19½ inches; of abdomen 20 inches.

Lungs: Over the entire anterior and posterior surface of the chest there were many loud, moist râles; there was no dulness or increase in the breath or voice sound.

Heart: On inspection there was a vigorous heaving systolic impulse in the right axillary line and in the epigastrium. On palpation, there was a strong, marked, systolic thrill felt over the entire precordium, but most marked and roughest at the lower end of the sternum and in the epigastrium. The apex beat was felt most prominently 6.5 cm. to the right of the midsternal line; on percussion, the left border was at the left sternal margin.

The right border in the sixth space was 9 cm., in the fifth space 8 cm., in the fourth space 7 cm., in the third space 5 cm., and in the second space 3 cm. to the right of the midsternal line. The nipple was 6 cm. to the right of the same line.

Corresponding to the area of palpable thrill there was a loud, rough systolic murmur. This murmur, less loud and less distinct, could be heard over the entire chest, anteriorly and posteriorly. It could also be heard in the great vessels of the neck. There was no Gerhardt's area of dulness in the second and third right interspaces. The pulse was 108 per minute; respiration, 28; abdomen, distended; liver and spleen in normal position, but somewhat enlarged, the liver being 7 cm. below the costal margin. Roentgenographic examination of patient Jan. 6, 1915, revealed the following findings:[1]

"The bony structures were first examined and it was found that except for some anomalous changes in the metacarpals, the osseous structure seemed normal. The left metacarpals (Fig. 2) when compared with the right were found to be short, thickened and club shaped, although the natural bone structure seemed to be normal.

"Taking up the thorax next, we found that we were dealing with a case of pure dextrocardia (Fig. 3). What under normal conditions would be considered the left heart was markedly enlarged and occupied at least two-thirds of the right chest cavity.

"Further investigation particularly to ascertain the position of the gastrointestinal tract, revealed the stomach in its normal position (Fig. 4) under the left diaphragm with the liver under the right. The injection of a bismuth solution into the colon per rectum gave us normal observation as to the location and size of the large intestine. The marked contraction seen in the middle third of the stomach we believe to be due to spasm.

---

1. Since the preparation of this report the patient died, but no postmortem examination was obtainable.

Fig. 1.—Patient's hands. Note clubbing of fingers.

Fig. 2.—Changes in bones of hand are clearly shown.

"Conclusions: The case is then one of pure dextrocardia in a child with a tendency toward anomalous development, which is shown by malformation of the metacarpals."

In addition to the physical signs already described, diagnostic data were also furnished by careful fluoroscopic study of the heart and an orthodiascopic tracing of its outline. By fluoroscopy, a distinctly

Fig. 3.—Roentgenogram showing dextrocardia.

enlarged and vigorous pulsating aorta was visible (Fig. 5). Beneath it, the pulsating area usually occupied by the pulmonary artery was not visible. In its stead, there was found a hugely dilated, prominent and "knob-like" left[2] auricle. The "left" ventricular shadow was also very much enlarged; its area could not be definitely distinguished from the "left" auricle, though their non-synchronous pulsations could

2. In this description in order to avoid confusion "left" and "right" are used in the sense as usually applied to the normal heart in its normal position, that is, "left" will refer to the larger and "right" to the smaller cavities of the heart.

be readily determined. The "right" auricle was enlarged. Beneath it, the beginning of the "right" ventricular shadow was seen deeply depressing the diaphragm during inspiration; from this it seemed probable that the entire "right" ventricle was enlarged. A comparison with the orthodiascopic tracing of a case of dextrocardia with no congenital defects (Fig. 6) and with normal cardiac outlines will serve to bring

Figure 4.—Roentgenogram showing stomach in normal position.

into sharper contrast the abnormalities described. The deviations in the first lead (the symmetrical lead) of the electrocardiogram (Fig. 7*b*) were all downward, an infallible electrocardiographic evidence of congenital dextrocardia (Neuhof[148]). Unfortunately, the second and third leads, though taken and studied, were subsequently lost. They showed runs of paroxysmal auricular tachycardia lasting several or many beats, with very much shortened auriculoventricular conduction time; the usual "onset" of paroxysmal tachycardia—an initial beat appearing after a compensatory pause (Lewis) was absent; in a

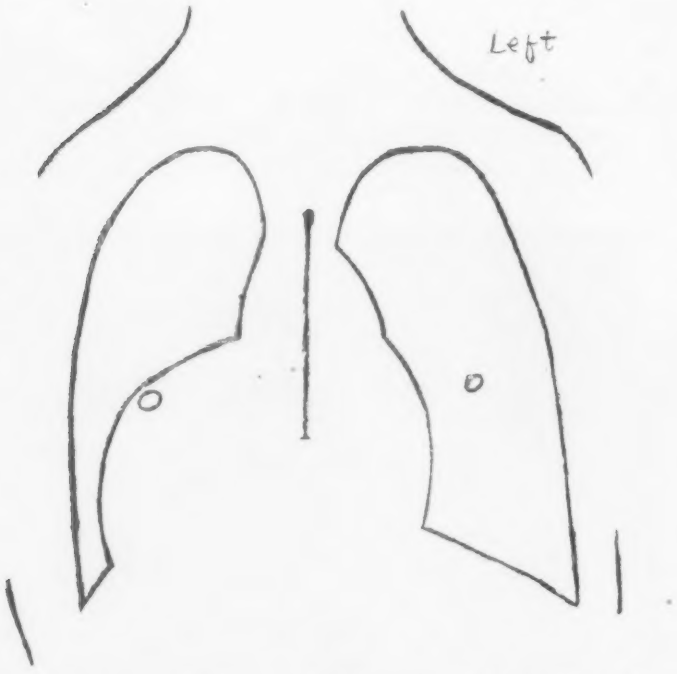

Fig. 5.—Orthodiascopic tracing of the patient with dextrocardia.

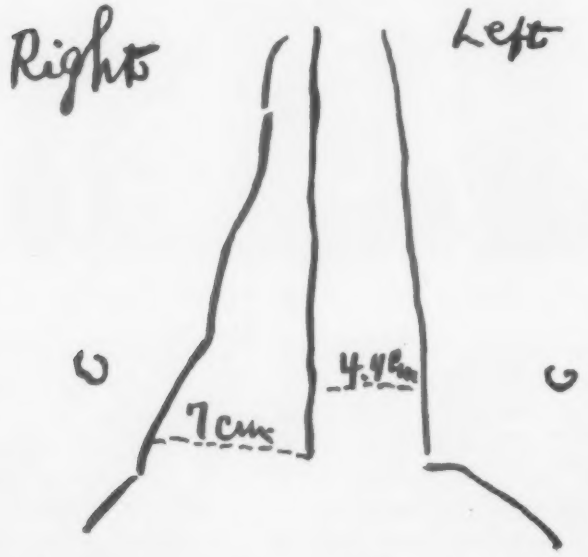

Fig. 6.—Orthodiascopic tracing of a case of dextrocardia with a normal heart for comparison with Fig. 5.

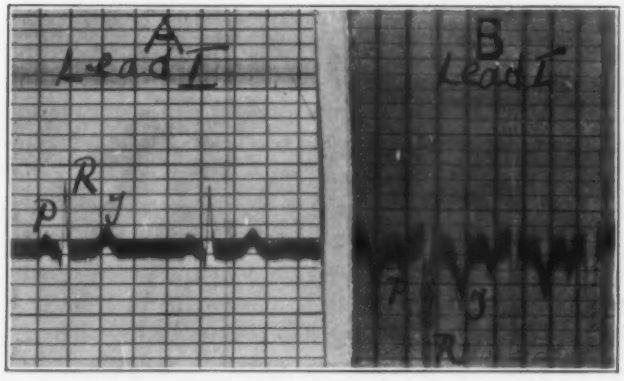

Fig. 7.—*A*, electrocardiogram (Lead 1) of patient with heart in normal position. The deviations P, R and T are positive. *B*, electrocardiogram (Lead 1) of the patient, D. L. The deviations P, R and T are negative. P is the auricular, R and T the ventricular complex.

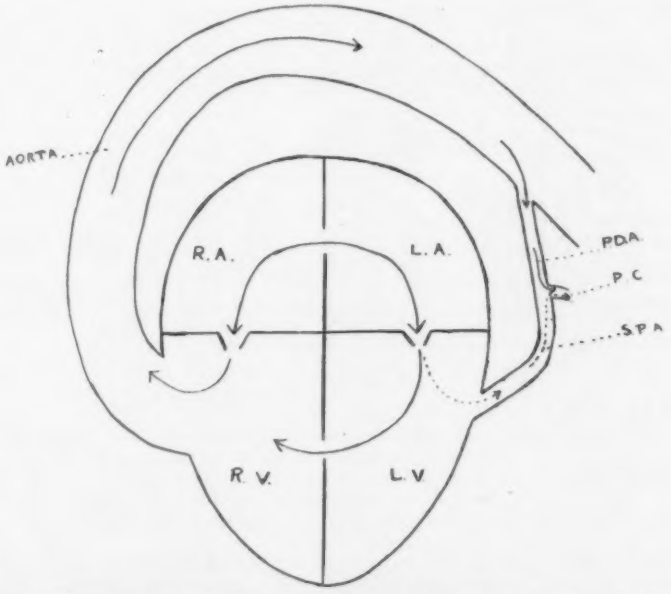

Fig. 8.—Diagram of probable cardiac circulation in the patient D. L. Solid arrows, main circulation; dotted arrows, minor circulation; RA, right auricle; RV, right ventricle; LA, left auricle; LV, left ventricle; PDA, patent ductus arteriosus; PC, pulmonary circulation; SPA, stenosed pulmonary artery.

section of the tracing, the normal rhythm was preceded by a compensatory pause (offset). A large deviation (the representative of an auricular beat) was present in Lead 2; this is usually regarded as evidence of "left" auricular hypertrophy.

A correlation of the physical and fluoroscopic signs—thrill and murmur over the entire precordium but most prominent at the xiphoid; apparent absence of the radiographic shadow of the pulmonary artery; an enlarged "left" auricle; an enlarged "right " auricle, "left" and probably also "right" ventricle point toward patent foramen ovale and interventricular septum as the probable congenital lesions. Orthodiascopic evidence of a patent ductus arteriosis was not found, but if the diagnosis of septal defects and pulmonary stenosis be correct, it is safe to assume that a patent duct was present; otherwise there would be no outlet for the blood necessary for pulmonary circulation, as evidenced by the diagram (Fig. 8) representing the probable directions of the blood current. These anomalies—pulmonary stenosis and patent ductus arteriosis—are not infrequently associated with septal defects (Carpenter,[150] von Rokitansky[151]). In a postmortem specimen of a heart normally placed in the chest (examined through the courtesy of Dr. B. S. Oppenheimer) septal defects and anomalies like those described were present; the ductus arteriosis was short, not dilated, and was surrounded by adhesions. If similarly distorted in our case, it would sufficiently account for the fact that it was not discovered during fluoroscopy.[3]

830 Park Avenue—1275 Madison Avenue.

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The following is a collection of cases cited from the literature, complete so far as possible from the year 1649. The cases given are of pure dextrocardia. Cases in which the liver and spleen were in abnormal position are not reported.

Lit. No.	Case No.	Year	Reference
1	1	1649	Riolani: Anthropographia, cited by Otto as pure dextrocardia; not cited by other authors.
2	2	1671	Moellenbrock: Miscellanea Curiosa, necropsy report; cited by a number of authors as true dextrocardia; Foggie says that the stomach also lay a little to the right.
3	..	1671	Hoffman: Cardianstrophe admiranda seu cordis inversio. Cited by Otto as true dextrocardia; Krieger states that this is the same case as Moellenbrock's.
4	3	1769	Eschenbach: Observata anatom. chir. med. rariora. One case found at necropsy; also cited by Krieger and Vehsemeyer.

3. Roentgenographic examination by Dr. William H. Stewart of the German Hospital and Dispensary, whom we desire to thank for his examination.

Lit. No.	Case No.	Year	References
5	4	1793	Abernethy: Tr. Philosoph. Soc., London, p. 59. One case cited by a number of authors as true dextrocardia; Foggie states that the liver lay in the middle line.
	5		
6	6	1816	Otto: Seltene Beob. z. Anat., Physiol. u. Path. Two cases, one found during life and confirmed at necropsy; one necropsy of a child. See also 1829, Otto.
7	7	1824	Würsburger Pathologisches Institut, necropsy, also cited by Mosler, 1866, and a number of other authors.
8	8	1826	Meckel: Archiv. f. path. Anat. u. Physiol., p. 41. One case in a premature child with complete absence of the right lung. Cited by a number of authors.
	9		
9	10	1826	Breschet: Repert. gen. d'Anat., 2:1. Gives four cases, only two of which are accepted by Schroetter, 1887, and three by Krieger.
10	11	1829	Otto: Neue seltene Beobacht. z. Anat., Physiol. u. Path. A third case. See Otto. 1816.
11	..	1832	Geoffroy-St.-Hilaire: Histoire gén. et partic. d. Anom. d. l'Organisation. Cites no new case; reviews those of other authors cited above.
12	..	1834	Lepington: London Med. Gaz., xv, 228. Heart on right side, serous effusion in lungs; gall-bladder absent. Dextrocardia possibly congenital, author does not state.
13	..	1836	Cooper: London Med. Gaz., xviii, 600. Dextrocardia with malformations of the thoracic viscera, cited by several authors as congenital, although Foggie states that it was a sequel to a right-side pleurisy of fetal origin.
14	12	1849	Cruveilheier, J.: Traité d'Anat. path. gen. One case of heart displaced to right by a diaphragmatic hernia.
15	13	1854	Stokes: Diseases of the Heart and Aorta. One case of heart displaced to right by a diaphragmatic hernia.
16	14	1857	Aertz: Bericht d. allg. Krankenhaus z. Wien. One case, cited by Vehsemeyer and Becker.
17	15	1858	Peacock: On Malformations of the Human Heart. Cited two cases, clinical diagnosis only, in which liver lay on left side; position of other organs not stated. Not cited by other authors.
	16		
18	17	1862	Maschka: Allg. Wien. med. Ztg., vii, 78. One case with patient's right lung absent; necropsy on newborn child; also cited by Foggie.
19	..	1863	MacLean: Lancet, London, 1863, ii, 159. Liver also transposed; position of other organs not stated.
20	18	1861	Jasinski: Wien. Med.-Haller, ii, 323. One case.
21	19	1866	Mosler: Berl. klin. Wchnschr., No. 21. Cites the Würzburger (1824) case; also one case of his own clinical diagnosis only; both cases accepted by Vehsemeyer and Becker, only the first by Lochte.
22	..	1869	Alvarenza: Jour. d. méd., de chir. et de pharmacol., Nos. 47 and 48. Not obtainable; according to title a case of "trochocardia."
23	20	1869	Bailey: Tr. Obst. Soc., x. Case with diaphragmatic hernia; also cited by Foggie.
24	21	1870	Schroetter: Jahrb. d. klin. Gesellsch. d. Aerzte, Parts 5 and 6; also in Med. Jahrb., xx, 189. One case which the author considers congenital with added traction from a diseased right lung. Also cited by other authors.
25	..	1871	Rodriguez: Gac. Med. d. Mex. Title reads "Transposition of Viscera," but the United States Surgeon-General's catalogue classifies it under displacements of the heart, and not under viscera; original not obtainable.
26	..	1873	Bienfait: Bull. Soc. Méd. d. Reims, p. 76. Title, "Dextrocardia," does not indicate whether congenital or not. Not obtainable.

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28	22	1877	Falck: Greifswald University Thesis. Title: Ein Fall von congenitaler Dextrocardia.
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31	25 26	1880	Krieger: Berlin University Thesis. Title: Zur Geschichte der congenitalen Dextrocardia. Cites one case from Leichenstern, not published elsewhere; also a case of his own, both congenital; cited by Foggie.
32	27	1881	Bramwell: Edinburgh Med. Jour., xxvii, 743. One case; also cited by Foggie.
33	..	1881	Robinson: Bull. New York Path. Soc., Series 2, i, 48. One case with cor biloculare and liver on left side; other organs normal; necropsy; also cited by Foggie.
34	28	1881	Ziemssen: Ann. d. stadt. allg. Krankenh. München. One case; also cited by Vehsemeyer and Becker.
35	29	1882	Pope: Lancet, London, 1882, ii, 9. One case, clinical diagnosis and necropsy; cited by a number of other authors.
36	30 31	1882	Wehn: Würzburg University Thesis. Title: Beitrage Zur Heterotaxie de Eingeweide. Includes two cases of true congenital dextrocardia, cited by a number of other authors.
37	..	1883	Küchenmeister: Die angeborene, vollstandige seitliche Verlagerung der Eingeweide. Cites no new cases; discusses the subject.
38	32	1887	Schroetter: Berl. klin. Wchnschr., xxiv, 448. One case of his own; also the one later described by Kundrat, 1888. Both cited by a number of other authors.
39	33	1887	Süssman: München. med. Wchnschr., xxxiv, 991; 1014. One case; cited by a number of other authors.
40	34	1888	Kundrat: Verhndl. d. Gesellsch. d. aerzte in Wien. One case; also described by Schroetter, and cited by a number of other authors.
41	35	1888	Gruss: Wien. med. Blätt., p. 202; also Berl. klin. Wchnscr., No. 8. One case, considered congenital by author; see also Pal, 1907, cited by a number of other authors.
42	36	1888	Bamberger: Wien. med. Blätt., p. 205. One case, considered congenital by author; later described by Pascheles, 1897, and Paltauf, 1901.
43	37	1888	Anselm: Riv. veneta d. Sc. Med., viii, 555. One case.
44	38	1888	Chabrely: Jour. d. méd. d. Bordeaux, xviii, 16. One case with both lungs diseased, no attack of pleurisy; lung disease possible cause of dextrocardia, but author considers it probably congenital.
45	39	1888	Michel: Med. Rec., xxxiv, 479. One case.
46	40	1889	Grünfeld: Prag. med. Wchnschr., xiv, 2. One case; also cited by other authors.
47	41	1889	Reddingius: Nederlandsch Tijdschr. v. Geneesk., No. 15. One case, necropsy; also cited by Vehsemeyer and Becker.
48	..	1890	Holt: Arch. Pediat., vii, 81. One case with enlarged right ventricle believed by author to be the cause of the displacement.

Lit. No.	Case No.	Year	References
49	42	1890	Grunmach: Berl. klin. Wchnschr., xxvii, 22. One case; pulmonary stenosis. Cited by a number of other authors as true dextrocardia.
50	..	1890	Niesel: Deutsch. med Wchnschr., xvi, 499. One case with a right-side pleurisy, not certainly congenital, but probably so; regarded by Vehsemeyer as congenital.
51	43	1890	Adie: Indian Med. Gaz., xxv, 117. One case, clinical diagnosis, persistent cough, but no symptoms of lung disease severe enough to cause the dextrocardia; probably congenital.
52	44	1890	Graanboom: Ztschr. f. klin. Med., xviii, 185. One case; also cited by a number of other authors.
53	45	1890	Cipriani: Lo Sperimentale, lxvii, 127. One case, clinical diagnosis and necropsy; also cited by Foggie.
54	46	1890	Sandhop: Greifswald University Thesis. Title: Ein Fall von kongenitaler Dextrokardie. One case; also cited by Nagel.
55	47	1891	Arnaud: Compt. rend. Soc. de biol., Series 9, iii, 4. One case.
56	..	1891	Inches: Maritime Med. News, iii, 63. One case; author doubtful whether congenital or caused by lung disease.
57	48	1891	Schott: Therap. Monatsh., v, 270. One case; also cited by a number of others; clinical diagnosis only.
58	..	1891	Varsi: An. Asist. púb., Buenos Aires, ii, 46. Not obtainable. Title: "A Case of Dextrocardia." Does not indicate whether congenital or not.
59	49	1891	Becker: Jena University Thesis. Title: Ueber Dextrocardia. One case; also cited by Nagel and Foggie as true dextrocardia.
60	50	1891	Hawkins: Tr. Clin. Soc., London, xxiv, 250. One case with pulmonary stenosis, but considered congenital by author; also by Foggie.
61	..	1891	Ewart and Bennett: Tr. Med. Soc., London, xiv, 438. One case, doubtful whether congenital or due to contraction of right lung.
62	51	1892	Heimann: Berl. klin. Wchnschr., xxix, 188. One case showing misshapen chest and other congenital faults; also cited by Foggie.
63	52	1892	Berwald: Berl. klin. Wchnschr., xxix, 1022. One case, clinical diagnosis and necropsy; also cited by Foggie.
64	53	1892-93	Bard: Lyon méd., lxxi, 583; lxxii, 15. One case with mitral stenosis; clinical diagnosis; also cited by Foggie and Loewenthal.
65	54	1894	Lochte: Beitr. z. path. Anat. u. z. allg. Path, xvi, 189. One case found at necropsy; also cited by Foggie.
66	55	1894	Stren: Norsk Mag. f. Laegevidensk, 4 R., ix, 93. One case; cited also by Vehsemeyer.
67	56	1894	Perregaux and Morestin: Bull. Soc. anat. d. Paris, lxiv, 968; 983. One case, clinical diagnosis and necropsy; right lung flattened and misplaced; also cited by Foggie.
68	57	1894	Droog: Nederl. Tijdschr. v. Geneesk., 2 R., xxx, Part 1, 872. One case; also cited by Loewenthal.
69	..	1894	Kreisch: Bonn University Thesis. Title: Ein Fall von hoch gradiger Verlagerung des Herzens in die rechte Seite. One case with enlargement of the left lung which may have caused the dextrocardia.
70	58	1894	Schroetter: Wien. med. Presse, Nos. 1 and 2. One case also cited by Lowenthal.
71	59	1894	Schmidt-Monnard: München. med. Wchnschr., xli, 584. One case; also cited by Loewenthal.
72	60	1895	Campbell: Montreal Med. Jour., xxiv, 515. One case, probably congenital; deformity of right chest caused by accident in childhood.

Lit. No.	Case No.	Year	References
73	61	1895	Goryanski: Boln. Gaz. Botkina, iii, 369. One case, probably congenital.
74	62	1896	Gerard: Lancet, London, Vol. 1 for 1896. One case; also cited by Loewenthal and Foggie.
75	63	1896	MacLennan: Brit. Med. Jour., Vol. 2 for 1896, p. 1314. One case; also cited by Loewenthal and Foggie.
76	..	1896	Fernet: Bull. et mém. Soc. méd. d. hôp. d. Paris, Series 3, xiii, 873. One case probably due to antenatal pleural disease, or disease in early infancy.
77	64	1896	Berks: Wien. klin. Rundschau., x, 497. One case; also cited by Foggie.
78	65	1896	Bramwell: Atlas of Clinical Medicine, iii, 116. One case with pulmonary stenosis, probably congenital; also cited by Foggie.
79	..	1896	Sobierajczyk: Berlin University Thesis. Title: Zur Casuistik der Dextrocardie. Same case as described by Vehsemeyer, 1897.
80	66	1897	Vehsemeyer: Deutsch. med. Wchnschr., xxiii, 180. Case diagnosed by Roentgen ray; also cited by others.
81	67	1897	Wardrop-Griffith: Brit. Med. Jour., Vol. 1 for 1897, p. 1287. One case considered congenital, with pulmonary stenosis.
82	..	1897	Pascheles and Paltauf: Wien. klin. Rundschau., xi, 473. Later report on Bamberger's (1888) case. See also Paltauf 1901.
83	68	1897	Auché and Bouyer. Jour. d. méd. d. Bordeaux, xxvii, 413. One case.
84	69	1898	Petit and Ravaud: Bull. et mém. Soc. méd. d. hôp. de Paris, Series 3, xv, 195; also in Gaz. d. Hôp., lxxi, 293. One case; also cited by Foggie.
85	70	1898	Berend: Gyermekgyogyaszat, p. 16. One case.
86	71	1898	Kroenig: Berl. klin. Wchnschr., pp. 230, 466. One case, clinical report and necropsy, with abnormal breadth of left lung; regarded by author as congenital; also cited by Schwalbe and Loewenthal; questioned by Foggie.
87	72	1898	Middleton: Glasgow Med. Jour., i, 244. One case with other congenital deformities, but no other viscera transposed; also cited by Foggie.
88	73	1899	Geipel: Festschr. z. Feier d. 50 jähr. Bestehens d. Stadtkrankenhaus z. Dresden-Friedrichstadt, p. 373. One case, clinical diagnosis and necropsy; also cited by Nagel.
89	74	1899	Leo: Jahrb. f. Kinderh., new series, l, 427. One case; also cited by Foggie.
90	75	1900	Bonheim: Kiel University Thesis. Title: Ueber Dextrocardia. One case, with other congenital defects; also cited by Foggie and Schwalbe.
91	76	1900	Crispino: Riforma med., xvi, Part 3, 436, 447, 459. One case, clinical diagnosis; also cited by Foggie.
92	77	1900	Schmilinsky: Deutsch. med. Wchnschr., xxvi Ver. Beil., 194. One case, clinical diagnosis; also cited by Foggie and Nagel.
93	78	1900	Chapman: Intercolonial Med. Jour., v, 309. One case; also cited by Foggie.
94	79	1900	Loewenthal: Ztschr. f. klin. Med., xli, 170. One case, clinical diagnosis and necropsy; also cited one case reported by Senator in 1899 (reference not given), clinical diagnosis by Roentgen ray.
95	80	1901	Monks: Brit. Med. Jour., Vol. 1 for 1901, p. 514. One case; also cited by Foggie.
96	81	1901	Baudouin: Gaz. méd. d. Paris, Series 12, i, 33. Cites no new case; discusses theory.

Lit. No.	Case No.	Year	References
97	82	1901	Chapot-Prevost: Compt. rend. Acad. d. sc., cxxxii, 223. Congenital dextrocardia in one member of a twin monster who lived after operation for separation.
98	83	1901	Paltauf: Wien. klin. Wchnschr., xiv, 1032. Postmortem of case reported by Bamberger in 1888 and Pascheles, 1897. Shows pericarditis which Paltauf considers the cause of the dextrocardia.
99	84	1901	Weinberger: Wien. klin. Wchnschr., xiv, 129. One case regarded by author as probably congenital, with symptoms of lung disease, also possibly congenital; cited by Foggie as truly congenital.
100	85	1901	Gossage: Tr. Clin. Soc. London, xxxiv, 220. One case reported by authors as congenital.
101	86	1902	Darnall: Med. News, lxxx, 446. One case.
102	...	1902	Alaux: Lyon University Thesis. Not obtainable; case not described elsewhere.
103	87	1902	Brudzinski: Gaz. lek. (Warsaw). One case.
104	88	1902	Baumgarth: Halle University Thesis. Title: Cor biloculare mit Dextrokardie. One case; also cited by Foggie and Nagel. Clinical report and necropsy.
105	89	1902	Wagner: Rostock University Thesis. Title: Zur Kenntnis der erworbenen und angeborenen Rechtslage des Herzen. One case, with facial defect also. Cited by Foggie.
106	..	1903	Beclére: Bull. et mém. Soc. méd. d. hôp. de Paris, Series 3, xx, 559. No case report, discusses diagnosis, chiefly of acquired dextrocardia.
107	90	1903	Wendling: Med. Blätt., xxvi, 563. One case, clinical diagnosis and necropsy; also cited by Foggie.
108	91	1903	Parkinson: Rep. Soc. Study Dis. Child., iii, 213. One case.
109	92	1903	Benfey: Berlin University Thesis. Title: Beiträge zur Lehre des Situs Transversus Partialsis. One case with pulmonary stenosis, also congenital.
110	93	1903	Flatau: Neurol. Centralbl., xxii, 643. One case; also cited by Foggie.
111	94	1903	Wright and Drake: Tr. Assn. Am. Phys., xviii, 272. One case with heart malformation, necropsy; also cited by Schwalbe.
112	95	1904	Carpenter: Brit. Jour. Child Dis., i, 160. One case, possibly congenital, possibly caused bv large cystic left lung; also cited by Foggie.
113	96	1904	Stone: Boston Med. and Surg. Jour., cl, 29. One case, probably congenital.
114	97	1905	Hoffman: Cor.-Bl. f. schweiz. Aerzte, xxxv, 111. One case, Roentgen-ray diagnosis.
115	98	1905	McIver: Charlotte (N. C.) Med. Jour., xxvii, 42. One case.
116	99	1906	Graham: Royal Army Med. Corps Jour., vii, 498. One case, possibly caused by pulmonary disease.
117	100	1906	Hochsinger: Deutsch. med. Wchnschr., xxxii, 1607. One case with heart defects, Roentgen-ray diagnosis.
118	101	1906	Neumann: Marburg University Thesis. Title: Ueber ein Fall von Dextrocardie. One case, clinical diagnosis; also cited by Nagel and Foggie.
119	102	1906	Potter, G. W.: Congenital Malformation of Heart, with Malposition of Certain Viscera and Absence of Spleen, Jour. Am. Med. Assn., Aug. 4, 1906, p. 363. One case, with stomach and intestines in the thorax; spleen absent. Congenital.
120	...	1907	Pal: Wien. med. Presse, xlviii, 809. Necropsy confirming diagnosis of case reported by Gruss in 1888.

Lit. No.	Case No.	Year	References
121	103 104 105 106	1907	Aufdermauer: Zurich University Thesis. Title: Ueber Dextrocardia congenita et acquisita. Reports five cases of congenital dextrocardia, one with right hydrothorax, clinical diagnosis; also cites one necropsy report by Lucchi.
122	107	1907	Tate: Jour. Royal Army Med. Corps, viii, 434. One case Roentgen-ray diagnosis; also cited by Foggie.
123	108	1907	Doolittle: Boston Med. and Surg. Jour., clvii, 662. One case.
124	109	1907	Hawthorn: Brit. Med. Jour., Vol. 1 for 1907, p. 1186. One case with valvular disease, clinical report.
125	110	1909	Schelenz: Berl. klin. Wchnschr., xlvi, 788; 840. One case, liver also transposed, other viscera normal.
126	111	1909	Carpenter: Rep. Soc. Study Dis. Child., p. 163; also in Brit. Jour. Child. Dis., vi, 444. Reports briefly one new case and refers to his 1904 case.
127	112	1909	Scandola: Gaz. d. osp. Milan, xxx, 1065. One case.
128	113	1909	Nagel: Deutsch. Arch. f. klin. Med., xcvi, 552. One case, clinical diagnosis and necropsy; tabulates other cases.
129	114	1910	Fossier: New Orleans Med. Jour., lxiii, 542. One case in which the heart was displaced to right when patient was lying on right side (about 6 cm.), apparently congenital.
130	115	1910	Whyte: Brit. Med. Jour., Vol. 2 for 1910, p. 198. One case.
131	116	1910	Foggie: Edinburgh Med. Jour., v, 428. One case with cor biloculare, clinical diagnosis and necropsy; tabulates other cases.
132	...	1910	Schwalbe: Morphologie der Missbildungen des Menschen und der Tiere, Part 3, Section 2, p. 370. Cites no new cases; reviews others.
133	117	1911	Stoerk: Mitt. d. Gesellsch. f. inn. Med. u. Kinderh. One case.
134	...	1911	Host: Norsk Mag. f. Laegevidensk, 5 R., ix, 435. Not obtainable; title does not indicate whether congenital or not.
135	118	1911	Brooks: Med. Rec., lxxix, 962. One case.
136	119	1911	Geissler: Wien. klin. Rundschau, xxv, 565. One case.
137	120	1911	Vandervelde and Catineau: Jour. méd. d. Bordeaux. One case. See also Vandeput, 1912.
138	121	1912	Mohr: Berl. klin. Wchnschr; also in Münch. med. Wchnschr., No. 7, 387. One case.
139	122	1912	Benjamin: Verhandl. d. München. Gesellsch. f. Kinderheilk.; also in Jahrb. f. Kinderh., lxxv, 367. One case.
140	123	1912	Culcer-Petresco: Paris Thesis. Title: Quelques considérations à propos d'un cas nouveau de dextrocardie congénitale. One case with cardiac malformations, clinical diagnosis, Roentgen-ray. Reviews other cases.
141	...	1912	Bello Moraes: Med. Contemp. (Lisbon), xxx, 190. Not obtainable; title does not indicate whether congenital or not.
142	...	1912	Vandeput: Jour de méd. d. Bordeaux, xvii, 37. Roentgen-ray report of case of Vandervelde and Catineau, 1911.
143	124	1912	Berenguer: Semaine méd. (Buenos Aires), xix, Part 2, p. 549. One case with diaphragmatic hernia.
144	125	1913	Cade, Rebattu and Gras: Province Méd., xxiv, 206. One case, dextrocardia acquired in prenatal life.
145	...	1913	Curti: Gazz. internat. d. med., xvi, 947. Not obtainable. Title does not indicate whether congenital or not.
146	126	1913	Giovannini: Policlin. 20: sez. Prat. 1479. One case.

Lit. No.	Case No.	Year	References
147	...	1913	Hermanides: Nederlandsch. Tijdschr. v. Geneesk., i, 1471. Not obtainable. Title does not indicate whether congenital or not.
148	...	1913	Neuhof: Post-Grad., xxviii, 262; A Case of Congenital Familial Dextrocardia, Jour. Am. Med. Assn., April 5, 1913, p. 1064. Case with patient's liver and spleen also transposed. (Roentgen-ray and electrocardiographic diagnosis.)
149	...	1915	Abbott: Osler's Modern Medicine, ed. 2, iv, 323. Cites no new cases, reviews others.
150	...	1894	Carpenter: Congenital Affections of the Heart, London, p. 16.
151	...	1875	Von Rokitansky: Die defecte der Scheidewande des herzen. Wien, vi, p. 8.

PATHOLOGY OF MYATONIA CONGENITA (OPPENHEIM)

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Complete postmortems in cases of this condition have been reported by Spiller, Baudouin, Lereboullet-Baudouin, Collier-Holmes, Rothmann, Beevor and Kaumheimer; Reyher-Helmholz in one necropsy examined only the musculature, and Bing, Collier-Holmes and Skoog respectively, examined a small piece of muscle excised from the patient during life.

The scarcity of the anatomic examinations recorded, especially in the American literature, the only reports given being those by Spiller-Smith and Skoog, justifies the publication here of the results of the examination of pieces of muscle and nerve in the case of myatonia congenita, which I reported in the AMERICAN JOURNAL OF DISEASES OF CHILDREN, October, 1914.

Sept. 8, 1914, the baby spoken of in that report became cyanotic while nursing and died suddenly, about one week after my last visit. A necropsy to examine the entire nervous system and the organs of inner secretion and to establish the cause of the sudden death (thymus, bronchitis?), was refused and I had to content myself with the excision of a large piece of the calf musculature throughout its depth, and of the peroneal and popliteal (tibial) nerves of the right leg, two hours after death.

The subcutaneous fat-tissue over the gastrocnemius muscle was about 8 mm. thick. The musculature was markedly pale pink, but apparently of undiminished volume. The specimens were preserved in a 10 per cent. formaldehyd solution and later were examined by Dr. Maximilian Herzog, pathologist of the Cook County Hospital, to whom I am indebted for the following report:

From the muscle removed pieces were embedded in paraffin, sectioned and stained with hematoxylin-eosin. The sections reveal a very advanced and marked condition of atrophy of muscular tissue (Fig. 1). The individual bundles of muscle fibers are small and separated from each other by a large amount of areolar fatty tissue. The latter presents the usual appearance of fat cells. It appears that from one-third to one-half of the entire original muscle tissue is now composed of areolar tissue. The latter appears to be quite vascular. Examination even under low power already shows enormous increase of sarcolemma-nuclei.

Under oil-immersion magnification it is seen that the muscle fibers left do not show well the normal striation; it is particularly the longitudinal striation

which is indistinct. On the other hand, the transverse striation in some places at least, is unusually distinct and it appears that the dark lines between the disks are widened (Fig. 2).

The muscle fibers are in cross-sections narrow, round or oval and vary in size (Fig. 3). No hypertrophic fibers are present. The vessels in the interstitial and areolar connective tissue appear to be numerically increased and perhaps enlarged in size, but there is no marked thickening of their walls. The sarcoplasma-nuclei under the high power show oval or round with a considerable amount of chromatic substance. In some places indications of karyokinetic figures can be seen. It is very evident that the great numbers of sarcolemma-nuclei and the karyokinetic figures indicate an attempt at regeneration or new formation. Of the sarcolemma-nuclei which are so numerously found in the

Fig. 1.—Muscle bundles small, separated by proliferated connective and by areolar fatty tissue; enormous increase of the number of sarcolemma-nuclei. From microphotograph, x 60.

sections, the majority have retained their normal relation directly under the sarcolemma itself, but here and there one sees places in the sections where the nuclei are found in the sarcoplasm itself, even in its very center. This of course is the condition which we generally see in regenerating voluntary muscle fibers. The development of the muscle appears to be retarded, presenting a prenatal stage, the not developed fibers being replaced by areolar tissue.

Peripheral nerves stained by Weigert's method, show marked, but not very profound degenerative changes. On the whole the myelin sheath is well preserved, but here and there it is thinned out and reduced to a very thin mantle of the axis-cylinder. One also occasionally sees breaks in the myelin sheath and the interspaces at the nodes of Ranvier are increased in size. There has

not been much proliferation of neurilemma nuclei, but the ordinary connective tissue between the individual bundles of nerve fibers is very much increased, so that the peroneal nerve contains more fibrous connective tissue than nerve tissue proper (Fig. 4).

Correlating the clinical findings with the pathologic findings described above, the profound atrophy and the decreased number of the muscle fibers explain the paralysis; the intense proliferation of the connective and fatty tissue, infiltrating and replacing the muscle substance, made the muscle appear of normal volume and concealed the

Fig. 2.—Transverse striation unusually distinct; atrophic fibers; no hypertrophic fibers. From microphotograph, x 1,000.

actual atrophy; the flabbiness of the musculature and the striking impossibility of distinguishing the muscle from the panniculus adiposus by palpation, as reported in the clinical history of the case, are the result of the same pathologic condition.

MUSCLE AND NERVE FINDINGS AS RECORDED IN LITERATURE

To gain a more comprehensive picture of the various findings in the muscles and the nervous system in this condition, so far as known, it is necessary briefly to review the literature on this subject.

In Spiller's case the muscles amid a hypertrophic interfibrillary and fatty tissue were little developed; the fibers were hyaline and very small; their longitudinal striation was indistinct, the number of the nuclei of the connective tissue considerably increased. The nervous system was intact.

Bing observed in a very small piece of muscle, excised from a 3½ year old child, only a certain increase of the number of the nuclei.

Skoog noted a great diminution in the size of all muscle fibers, their diameters measuring from 8 to 12 microns; their myoplasma in many

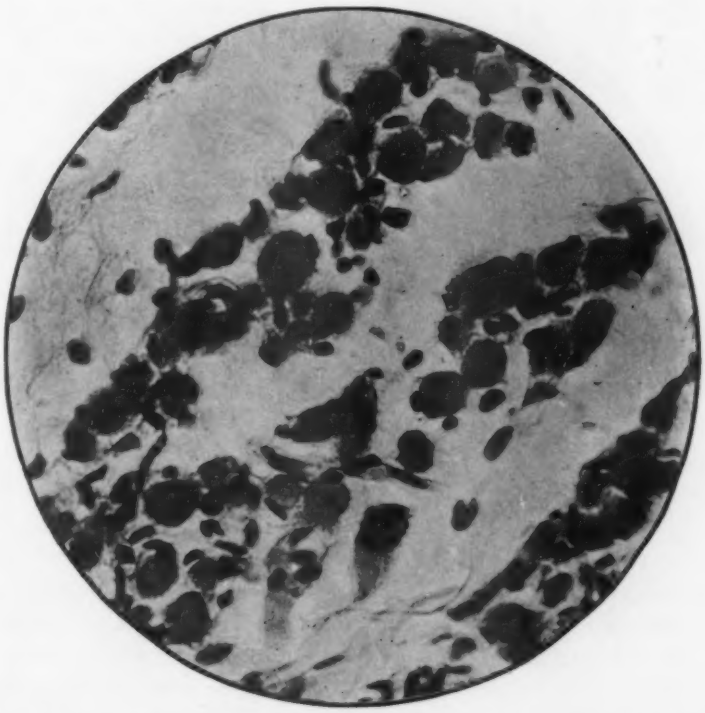

Fig. 3.—Atrophic muscle fibers of various diameter; fibers separated by proliferation of connective and fatty tissue. From microphotograph, x 450.

places had undergone a complete degeneration and replacement with adipose or connective tissue. There was, moreover, much fatty tissue between the individual bundles, a very marked proliferation of the sarcolemma nuclei and a pronounced thickening of the walls of the vessels, including the capillaries.

While in these cases, including my own, only atrophic fibers were found, other authors, as Reyher-Helmholz and Collier-Holmes have observed, in addition, hypertrophic fibers in such numbers that the

microscopic pictures resembled more the ordinary findings of progressive muscular dystrophy.

Reyher-Helmholz noted that some of the muscles of their patient, especially those of the lower extremities, were much affected. The fibers—of which some were slender, some hypertrophied—were scanty and interrupted by adipose and connective tissue which abounded in nuclei; also the transverse striation was indistinct or entirely absent. Unfortunately the central nervous system was not examined to

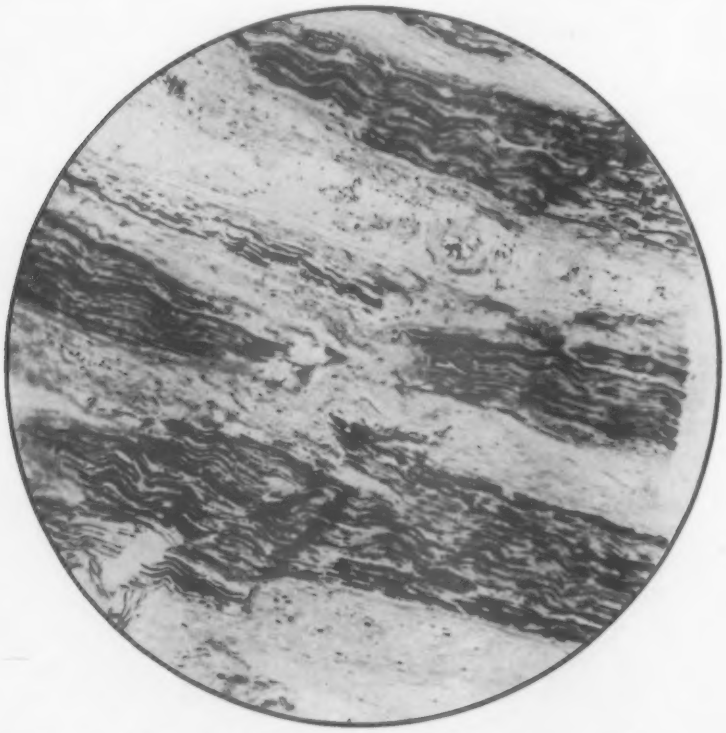

Fig. 4.—Peripheral nerve, stained by Weigert's method. Marked increase of fibrous connective tissue between the nerve bundles. From microphotograph, x 65.

determine whether these changes were primary in the muscles themselves or secondary to an affection of the anterior horns of the spinal cord.

Collier-Holmes, having excised a piece of the hamstring muscle (in Case 2) under general anesthesia, noted that macroscopically it was markedly pale and very much infiltrated by fat tissue. Microscopically only a few fiber bundles were found to be normal; a great number of the fibers were extremely small, with an average diameter

of from 6 to 12 microns; their contour was irregular, being principally oval or round, and never presenting the normal polygonal or facetted appearance of cross-sections. These small fibers, when cut longitudinally, however, appeared of fairly uniform caliber and had a well-preserved cross striation. In some of these there was marked proliferation of the sarcolemma nuclei. Very striking indeed was the presence of numerous enormous fibers with diameters of from 100 to 150 microns. These giant fibers here and there presented regressive changes; some of them contained central nuclei either embedded in normal myoplasma or surrounded by a zone of undifferentiated sarcoplasma. In other fibers the regressive changes had led to vacuolization and a splitting up, or the more or less disintegrated myoplasma was invaded by the sarcoplasm and sarcoplastic nuclei. The connective tissue was greatly increased and in places even penetrated between the individual muscle fibers. There was also a notable amount of loose adipose tissue between the muscle bundles. The muscle spindles on the whole proved to be normal, but in places their sheaths appeared slightly thickened. Considerable thickening of the walls of many blood vessels was noticeable. In the muscles were remarkably few myelinated nerves and the myelin sheaths seemed poorly developed. In Case 1 (complete necropsy) the muscles presented lesions similar to those just described.

The myelin sheaths of the peripheral nerves were imperfectly developed, the ventral spinal roots were slender, the number of their fibers reduced and the myelin defective. The great motor ganglion cells of the anterior horns were reduced in number and size and were abnormally angular and irregular in shape.

Baudouin also has described hypertrophic in addition to atrophic fibers, proliferation of the connective tissue, multiplication of sarcoplasma nuclei and indistinct transverse striation. These changes, however, were of minor degree. The nervous system revealed profound alterations. The size and perhaps also the number of the cells of the anterior horns, was diminshed; in the various nerve trunks many axis-cylinders were without myelin; the nuclei of the sixth and twelfth cranial nerves showed chromatolysis. Baudouin ascribed the condition to a retarded myelination of the nerves and an inhibition of the development of the cells of the anterior horns.

Rothmann's case presented even more extreme lesions. In addition to extensive atrophy of the great motor cells throughout the anterior horns of the cord were also alterations of the white substance and a diminution in number of the cells of Clark's columns. The smallness of the cord was striking. The motor ganglion cells of the hypoglossus nucleus were numerically decreased and showed chromatolysis. The

peripheral nerves were partly thickened, owing to enlarged septa of connective tissue, with multiplication of the nuclei. The transverse diameters of the nerve filaments were reduced, the single fibers were also very slender, though not changed in number or structure, and pursued a wavy course. The principal changes in the muscles consisted in pronounced proliferation of the intermuscular connective tissue, in abundant small cellular proliferation and marked development of fatty tissue between the muscle fibers, which last had partly disappeared.

Likewise Kaumheimer at postmortem in his case found, in addition to muscular changes, profound alterations of the nervous system. There were signs of severe degeneration of the peripheral nerves: marked diminution in number of the fibers, corresponding proliferation of the perineural and endoneural connective tissue and a marked increase of Schwann's nuclei. Many of the remaining fibers were small and of varying diameter; some showed a wavy course in longitudinal cuts. In various places the myelin sheaths and the axis cylinders were absent, though no marrowless axis cylinders could be detected.

The central nervous system showed extensive alterations, most pronounced in the motor cell columns of the spinal cord and in the medulla oblongata. There was proliferation of the glia in the marrow of the brain hemispheres, in the medulla oblongata and in the white substance of the spinal cord; atrophy in the nucleus of the hypoglossus nerve, in Clark's columns and the anterior horns of the entire spinal cord.

NATURE OF THE DISEASE

The views concerning the nature of the disease vary, inasmuch as many difficulties arise in the interpretation of the various pathologic changes.

Before any pathologic examinations were on record, Oppenheim regarded the disease as a retardation of development of the muscles, a conclusion later supported by Spiller, who failed to discover either gross or minute changes in the nervous system. Oppenheim, however, considered also the possibility of retarded development of certain nerve centers and their function, especially of the anterior horns of the spinal cord.

The findings of subsequent authors demonstrated indeed that the whole peripheral neuron may be affected, different segments varying in degree in different cases. If we remember that the ganglia cells of the spinal cord, the peripheral nerves and the muscle constitute a physiologic unit, this is not surprising.

The muscle findings place the affection near the group of the so-called primary myopathias—dystrophia musculorum progressiva. But while in myatonia the lesions extend to the nervous system in the majority of cases so far reported and are only exceptionally limited to the muscles, on the contrary, in dystrophy we observe, as a rule, muscular alterations alone, such as atrophy, hypertrophy of the fibers, multiplication of the muscle nuclei, etc., though in exceptional cases—those of long duration especially—degenerative alterations of the nerve elements were also found, these perhaps being secondary. (Transitional cases of Heubner, Struempell, Preis.)

On the other hand, since in primary spinal atrophies combinations with pseudohypertrophy of muscles also are known, the anatomic demarcation between myelopathic (spinal) and myopathic atrophies becomes still less sharp; though it must be admitted, that the hypertrophic fibers observed for instance in poliomyelitis acuta or in neurotic muscular atrophy were apparently present only in small number. All this tends to demonstrate the possibility of a relationship between primary dystrophy and spinal muscular atrophy.

A number of authors hold myatonia as at least a process closely related to muscular dystrophy, if not a congenital form of this disease. Jendrassik thinks that the possibility of improvement in myatonia as opposed to dystrophy forms no essential difference in the nature of these affections; for he considers in myatonia the clinical improvement not to be a real (anatomic) restoration but rather an improvement of the innervation of individual muscles or muscle fibers originally less affected. At present clinical evidence does not permit us to classify myatonia as progressive dystrophia musculorum. Also the frequency and degree of peripheral nerve and spinal cord alterations in myatonia evidence dissimilarity from dystrophy, and suggest a relationship to the spinal atrophies.

The possibility of the existence of primary lesions in the muscles in at least a number of cases will not be denied in the presence of such findings as Spiller reports though indeed the theory is permissible that in such cases the original minor affection of the motor ganglia cells was not irreparable, and for this reason the ganglia cells in the later stage of the process failed to show the former lesions. On the other hand, such a degree of lesions of the nervous system as quoted above seems almost to exclude the probability of anatomic restitution; and in view of my own findings I am inclined to attribute the clinical improvement in my case more to amelioration of the function than to a progressive anatomic development.

Rothmann calls attention to the great similarity of his findings in myatonia to those of Wernig-Hoffmann's spinal infantile atrophy of

the muscles, and also emphasizes that the clinical differences are not always marked, as cases of Wernig-Hoffmann's atrophy without family grouping are known, and, on the other hand, myatonia has been found among several members of the same family. (Sevestre, Collier-Wilson, Sorgente). Rothmann unites both forms in one group of spinal atrophies in spite of the clinical fact that myatonia is congenital and not progressive (with rare exceptions), whilst Wernig-Hoffmann's atrophy usually begins in the second or third semester of life. He thus distinguishes a congenital and an early infantile form which show no sharp line of demarcation. In his opinion, improvement in cases indicates that they should be regarded as of myatonia, while progressive cases belong to the Wernig-Hoffmann type of the disease.

He excludes fetal poliomyelitis in his case. Marchi specimens proved to him the fetal origin of the disease and that the degenerative processes were continued during the first months of life. He considers that agenesy of the ganglion cells is improbable.

As to the assumption that the disease shows a relationship to Moebius' infantile *Kernschwund* no verdict can be given at present; theoretically the possibility must be admitted that an affection does exist in which the cells of the anterior horns are aplastic or defective, a condition of similar nature to that shown in Moebius' *Kernschwund;* so that their function is either absent or is only present after a period of inactivity.

A resistentia minor of the protoneuron may be possible in all these affections owing to developmental inferiority, which is only temporary in some cases and to a certain extent reparable (myatonia), but which in others is progressive and lethal (spinal muscular atrophy).

Kaumheimer concludes from the histologic examination in his case that myatonia must be considered as either a toxic or endogenic affection; the theory that the pathologic basis of this disease lies in inhibition of development or inflammatory processes should be discarded. The process is not necessarily complete at birth, as alterations of the muscles may result even months later.

Berti suggested from his study of his own and other reported cases that a relationship exists between myatonia and subcretinism or submyxedema; and Cattaneo and Silvestri likewise believe the disease to be due to a disturbance of some internal secretion. The sclerotic changes of the thymus noted by Spiller-Smith and the sclerosis of the spleen, thyroid gland and thymus observed in Baudouin's case seemed to support these views. In Rothmann's case the thymus was enlarged, Hassal's corpuscles being strongly developed; though he did not regard this as important. Other authors, as Collier-Holmes, Lereboullet-Baudouin and Rothmann could find no changes in these organs, so that

these isolated positive findings hardly offer a sufficient basis for broad conclusions. The improvement which occurred in Silvestri's case on adrenalin administration and in Lepine's case after thyroid therapy, may have been accidental since Oppenheim's disease has a tendency toward spontaneous improvement.

Though our experience concerning the function of the thymus so far does not establish its relationship to the muscle and nervous lesions seen in Oppenheim's disease, we know, nevertheless, that thymectomy in animals causes myasthenia with certain neuromuscular alterations in addition to the well-known skeletal changes. These are, simple atrophy with transition to degenerative atrophy in the muscles, marked reduction in the caliber of the fibers, partial loss of the transverse striation, multiplication of the sarcolemmal nuclei and the nuclei of the internal perimysium, and distinct proliferation of the interfascicular connective tissue with formation of adipose tissue.

These results of animal experiments possess interest also in connection with the muscular lesions in man, found in myasthenia gravis and myasthenia pseudoparalytica with persistence of the thymus, and in connection with the degenerative changes of the muscles seen in disturbances of various other endocrine glands, as in Basedow's disease.

According to Biedl examination of the nervous system of thymectomized animals revealed only minor alterations in the peripheral nerves and slight degenerative conditions in the spinal cord. The myelin sheaths in the cord showed disintegration, the ganglion cells of the gray matter were swollen, lighter stained, with indistinct protoplasm and changes of the nuclei, indicating also a beginning disintegration in them. These observations although for our problem merely suggestive and of only speculative service, should induce us nevertheless to direct careful attention to the organs of internal secretion at necropsies in future in cases of Oppenheim's disease.

At present the morbid agent in the spinal and muscular changes, the cause of Oppenheim's myatonia in the fetus is not known, nor do the findings allow an undisputed classification of the disease.

MUSCULAR LESIONS IN RACHITIC MYOPATHY

It may not be amiss to call attention here to affections of the muscular system demonstrated in such general diseases as rachitis. This rachitic myopathy is considered a primary, specific and co-ordinate to the alterations of the skeleton; it bears a certain clinical resemblance to myatonia congenita. The morphologic changes found in the texture of the muscles in the clinically pronounced hypotonia and pseudoparesis of rachitic babies were marked reduction of the caliber of the

fibers, defective transversal striation, marked longitudinal striation, striking increase in the number of the muscle nuclei, lack of fatty tissue, indistinct sarcolemmal edges and densely crowded fibers, conditions different from those seen in Oppenheim's disease.

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A CASE OF CONGENITAL HEART DISEASE WITH DEMONSTRATION OF SPECIMEN *

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Eli S. was born at full term, after a normal delivery, about Jan. 1, 1906. He weighed 3 pounds. His mother noticed, when he was 4 months old, that his lips, cheeks and nails were always blue and that at times he was blue all over. He was feeble from the beginning and, when he was old enough to exert himself, showed dyspnea on exertion. He had scarlet fever when he was 2 and whooping-cough when he was 3 years old. He was brought to the Children's Hospital for the first time Dec. 1, 1909, when he was about 4 years old. At that time he was small, but well nourished. His lips and nails were very blue and the mucous membrane of the mouth was cyanotic. There was marked, general cyanosis when he cried. There was marked clubbing of the fingers and toes and some bulging of the precordia. The heart was somewhat enlarged in both directions, and a very faint murmur was heard at the junction of the sternum and the third and fourth ribs on the left. There was also a very slight systolic murmur in the pulmonic area. These observations were made by a house officer, however, so that there is some question as to their accuracy. The patient was seen and examined repeatedly by various members of the visiting staff from that time until his final illness and no murmurs were ever heard by any of them. He developed well, was active and showed no evidences of discomfort referable to his heart. He was generally cyanotic, and the clubbing of the fingers and toes became extreme. The conjunctivae were discolored. There was no enlargement of the liver or spleen.

The blood showed from 120 per cent. to 140 per cent. of hemoglobin by the Sahli apparatus. The red corpuscles varied between 7,800,000 and 13,675,000, most of the time being between 11,000,000 and 12,000,000. The white count varied between 8,000 and 12,000. The differential count of the white cells showed on the average 32 per cent. of mononuclears and 68 per cent. of polynuclear neutrophils. There were never any abnormalities in the red cells.

The urine was normal at repeated examinations.

Aug. 9, 1914, when a little more than 8½ years old, the patient had an acute attack in which he vomited blood and passed blood in the stools. He was brought into the hospital in collapse, but had no recurrence of the hemorrhages. The urine contained albumin, casts, and a little microscopic blood. It was clear in about a month. The heart at that time measured 4 by 9.5 cm. There were no murmurs. The liver was 1 cm. below the costal border. The spleen was not palpable. The blood showed a white count of 33,800 at that time.

Subjective symptoms developed for the first time in early November, 1914. The patient was able to walk, but any greater exertion caused cardiac distress. He was unable to attend school or play. He complained of pain in the right chest. The cardiac measurements were 4 by 9 cm. There were no murmurs. The liver was 1 cm. below the costal border. The spleen was not palpable. He was examined at this time by Dr. L. E. Holt, who also heard no murmur.

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* Read at the Annual Meeting of the American Pediatric Society, Lakewood, N. J., May 25-27, 1915. From the Children's Hospital, Boston.

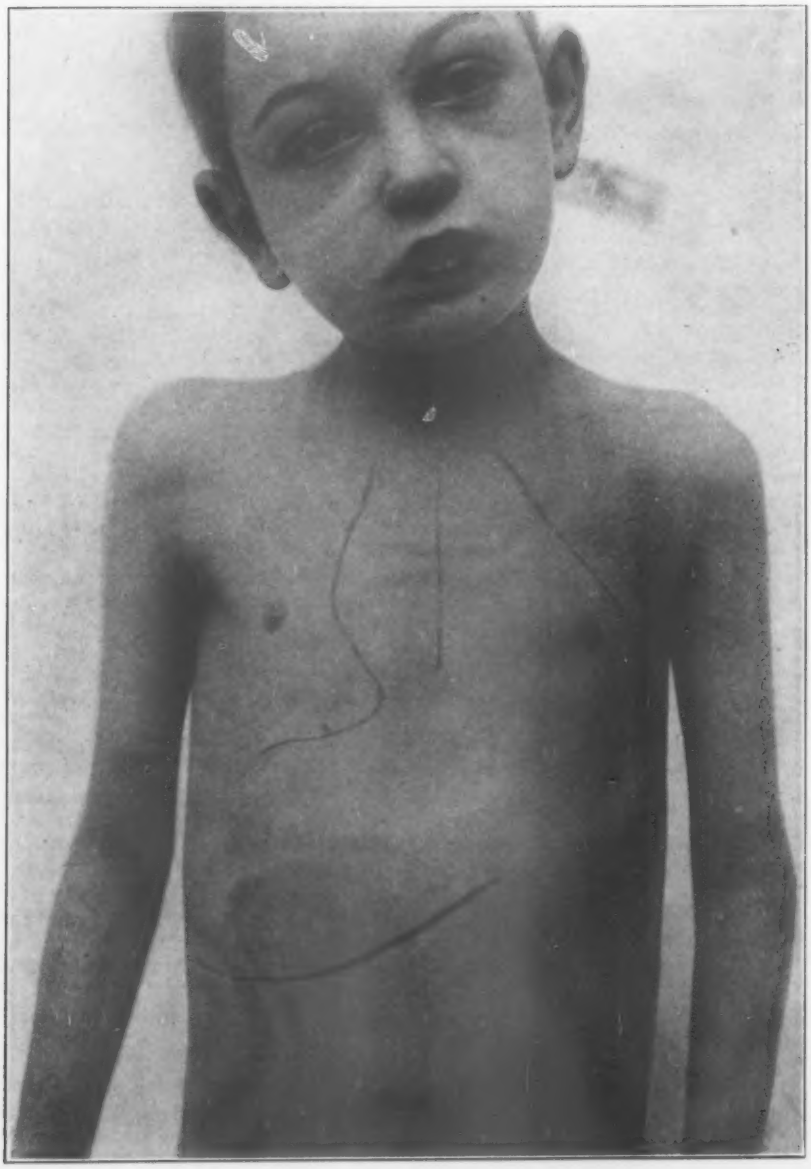

Fig. 1.—Patient with congenital heart lesion.

The patient stayed in the hospital only a few days at this time, but returned Nov. 30, 1914, with the story that he had been perfectly well until the night before, when he began to have constant and severe pain in the abdomen, with high fever and vomiting. The cyanosis was greater at this time than ever before and the temperature was 103 F. The condition of the heart was the same, except that it extended 10 cm. to the left. The urine was full of normal blood and also contained bile. The patient became slightly jaundiced during the next few days. He quickly improved, however, so that on December 4 the urine was normal. He was examined carefully December 2 by myself and several other men, including Dr. H. A. Christian. There were no murmurs

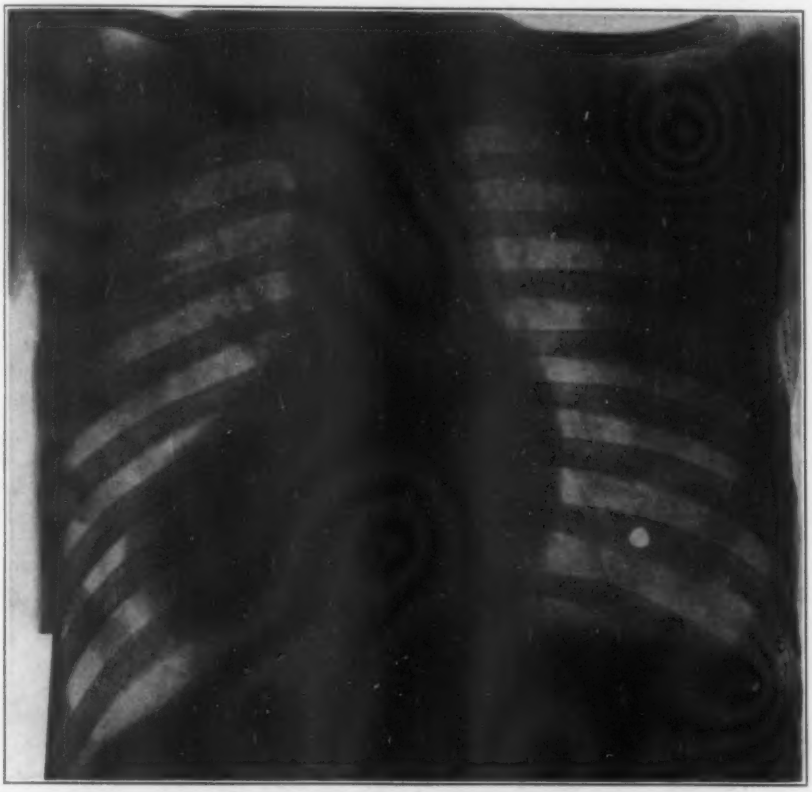

Fig. 2.—Heart as shown by Roentgen ray.

in the heart and there was no palpable thrill. The temperature had in the meantime dropped to normal in the morning, with an evening rise to 100 or 101 F.

He was not examined December 3. December 4, a diastolic murmur was present over the whole precordia, loudest in the pulmonic area.

The examination of the heart, Dec. 7, 1914, was as follows: The right border of the heart was 4.5 cm. to the right of the median line, and the left border 11 cm. to the left of the median line. The upper border was at the lower border of the second rib. The action was regular. There was a high-pitched murmur beginning just before the first sound, accompanying the first sound and continuing a little after it. The second sound was accompanied by a long, somewhat rough murmur, which was loudest in the third left space. It

was louder in the pulmonic area than in the aortic area or at the apex. The second sound was distinct. Figures 1 and 2 show the conditions at that time.

The measurements of the heart on the roentgenogram (Fig. 2) are as follows: Right border: first space 3 cm.; second space 4 cm.; third space 4.5 cm.; fourth space 2.5 cm. Left border: first space 3.5 cm.; second space 7 cm.; third space 11 cm.; fourth space 11.75 cm.

At this time the cyanosis was somewhat less than it had been in the past. There was no edema or ascites. The liver was palpable 3.5 cm. below the costal

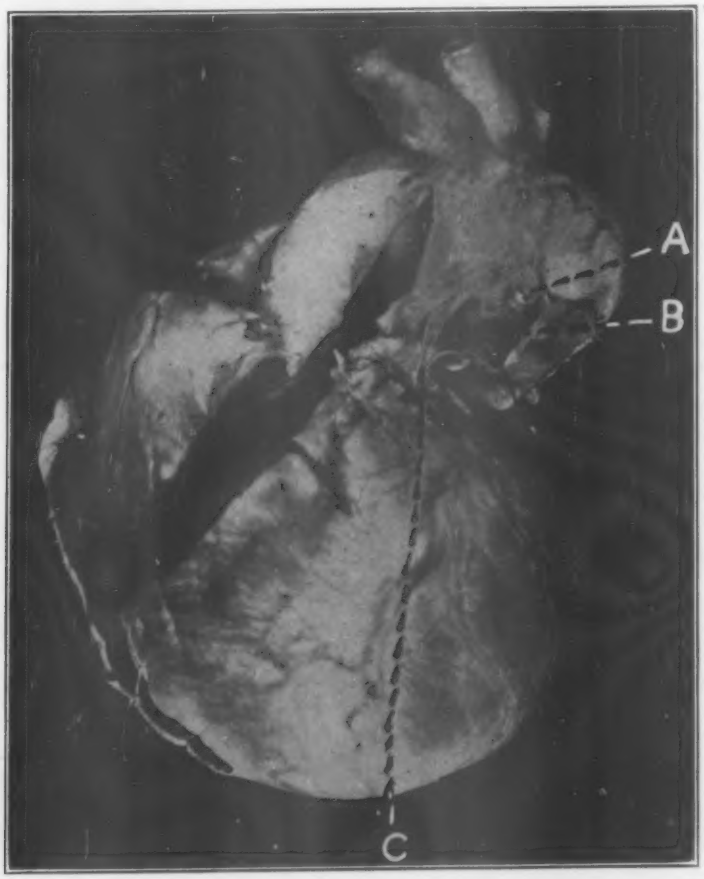

Fig. 3.—View of heart showing *A*, ductus arteriosus, *B*, two vessels to lungs, and *C*, rudimentary pulmonary artery.

border in the nipple line. The spleen was not palpable. The hemoglobin came down to between 100 per cent. and 115 per cent. by the Sahli apparatus. The red corpuscles ranged between nine and eleven millions and the white corpuscles between 9,000 and 10,000. The blood pressure, by the Tycos apparatus, was 120 in systole and 50 in diastole, giving a pulse pressure of 70.

The temperature rose again after the appearance of the murmur, but after a week dropped to normal in the morning with an evening exacerbation to from 100 to 101 F. By the middle of January the systolic murmur had almost disappeared and the diastolic murmur had become much fainter. The general condition continued unchanged, as did the condition of the heart. The patient died suddenly Feb. 10, 1915, presumably as the result of an embolus.

A positive diagnosis of congenital heart disease was made when he was first seen. The malformations with which the symptoms were reasonably consistent were absence of the ventricular septum, transposition or irregular origin of the great vessels and pulmonary atresia with some secondary malformation. There ought not to have been any second pulmonic sound, however, if there was pulmonic atresia, and it should have been accentuated if there was simply a transposition of the vessels. The most reasonable diagnosis, therefore, seemed to be absence of the ventricular septum. A single vessel coming from a single ventricle had been mentioned but had not been seriously considered.

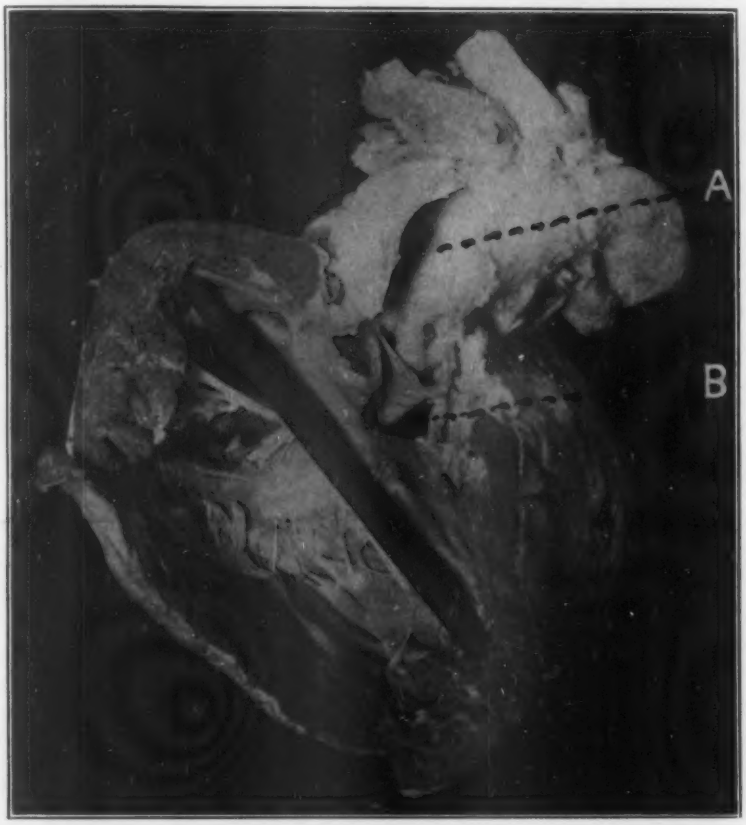

Fig. 4.—Heart cut open, showing *A*, single artery and *B*, opening between ventricles.

Unfortunately, it was possible to obtain only a partial necropsy. The heart and lungs alone were examined. The results of the examination by Dr. S. Burt Wolbach are as follows:

Gross anatomical diagnosis:

1. Complete congenital atresia of pulmonary artery.
2. Congenital defect of interventricular septum.
3. Persistent ductus arteriosus.
4. Acute vegetative endocarditis.
5. Thrombosis of ductus arteriosus.
6. Patent foramen ovale.
7. Dilatation and hypertrophy of right ventricle.

The heart is greatly enlarged and weighs 130 gm. (normal, 105 gm.). The general shape is that of a normal heart, but the right border is rounded and it is evident that the right ventricle is enlarged. The auricles are in their normal position. The appendage of the left auricle, however, is narrow and projects as a curved process, 2.5 cm. long and 0.5 cm. broad.

There is a single arterial trunk which arises at the base of the ventricles anterior to the auricles. The tips of the right and left auricular appendages appear on their respective sides of this vessel. On opening the heart it is found that this vessel arises from both ventricles. Its orifice is directly above the interventricular septum, which is deficient. This deficiency corresponds to that resulting from the absence of the membranous portion of the septum. Its upper border is the attachment of the anterior segment of the mitral valve. Its right pillar extends to the base of the right anterior segment of the semilunar valve of the arterial trunk, while its left pillar extends to the junction of the left anterior and the posterior segment of the valve. The size of the interventricular opening is 2 by 1 cm. (after hardening). The arterial trunk is apparently effectively guarded by the semilunar valve, a posterior and a right and left anterior. The free edges of these segments are slightly thickened and covered with pale, grayish, friable granulations. The coronary arteries take their origin behind the posterior and the left anterior segments. The size and shapes of the segments are those of a normal aorta in a heart of this size. The circumference is 8 cm. The arch corresponds to the arch of the aorta and is continuous with the descending aorta. The vessels given off are in the order named; the innominate, left common carotid, a small thin-walled ascending vessel and a left subclavian. At the location of the ductus arteriosus a large vessel takes origin by a short trunk, which divides into a right and left pulmonary artery. From this trunk running down along the main arterial trunk, is a thin-walled, small calibered vessel, 2 to 3 mm. in diameter. This vessel ends blindly, gradually disappearing to a point in the myocardium between the posterior and left anterior segments of the valves of the main arterial trunk.

The orifice of the pulmonary trunk into the arch of the main arterial trunk is 0.4 cm. in diameter, and nearly wholly occluded by a firm, grayish-red clot. This clot is cylindrical in shape, 0.4 cm. long, and attached firmly over one-half its circumference (organized).

The mitral and tricuspid valves are normal in appearance. The mitral measures 8.5 cm. in circumference at the attachments of the segments, and the tricuspid valve measures 10 cm.

The venous orifices in the auricles are normal in number and position. The foramen ovale is patent by an oblique passage.

The endocardium is normal, except for the aortic changes noted and a few minute vegetations on the inferior border of the opening between the two ventricles.

The myocardium is firm, of normal color and consistency. The left ventricular wall averages 1 cm. in thickness; the right is 0.9 cm.

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ACUTE INFECTIVE ENDOCARDITIS

DUE TO STREPTOCOCCUS ATTENUANS IN A CHILD ONE YEAR OLD *

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Acute endocarditis, simple or malignant, is (with rare exceptions) a secondary and not a primary process. The morbid condition is the result of an infection by some micro-organism or its toxin. The action, Anders believes, is assisted by the friction between the blood current and the surfaces of the valves. The pathologic processes, though identical in nature, differ in degree and severity in the so-called simple, the intermediate grades and the more rapidly fatal or ulcerative type.

A good working classification, either etiological, anatomical, or clinical, is not easy to make. According to the *nature of the infecting* agent we speak of streptococcic, staphylococcic, pneumococcic, rheumatic, typhoid or gonococcic; according to *the character of the lesion,* of verrucose or ulcerative; according *to the severity of the symptoms,* of benign and malignant varieties.[1]

Lamb and Paton[2] report a case of vegetative endocarditis caused by a heretofore undescribed spirillum (*Spirillum surati*).

Dean recorded an example of generalized actinomycotic infection in which the endocardium was implicated. The elaborate and thorough work of E. Libman in particular, has given us valuable information regarding the cases of subacute infective endocarditis.

The most frequent cause is acute articular rheumatism, comprising about 10 per cent. of the acute cases. In lobar pneumonia Osler found it in eleven out of twenty-three cases. Septic conditions may also act as a cause. It has occurred in measles, tonsillitis, scarlet fever, typhoid, erysipelas, chorea, tuberculosis, etc.

The protean aspects of the disease are influenced or rather due to the specific character of the infecting organism. The manifestations may be those of septicopyemia, in which chills, fever, hemorrhages, septic emboli, etc., are due to suppurative lesions. Or in the non-suppurative forms, the picture is that of a septicemia with high and irregular temperatures. The symptoms are so diverse that in many cases it is impossible to differentiate the suppurative from the non-suppura-

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* Read by title at the Annual Meeting of the American Pediatric Society, Lakewood, N. J., May 25-27, 1915.

1. Osler: Modern Medicine, 1908, iv, 133.
2. Lamb, Albert R., and Paton, F. Wade: THE ARCHIVES INT. MED., Sept., 1913, p. 259.

tive varieties. Not infrequently, when cerebral symptoms predominate, a lumbar puncture is advisable in order to exclude a possible meningitis. The cardiac symptoms vary. We may have extensive involvement of the valves with negative physical signs on auscultation. In other cases, more or less distinct murmurs, depending on the valves involved, may be discovered. As E. Libman remarks, "the absolute diagnosis rests on the cultural study of the blood."

In arriving at a diagnosis it is of the utmost importance to study the previous history and conditions under which the individual case occurs. With the symptoms referred to above and the evidences of emboli, petechiae, etc., a diagnosis is possible, even though cardiac murmurs are absent. Blood cultures, however, are necessary in order to establish the identity of the organism, the specific cause of the disease under consideration.

Streptococcic endocarditis is of frequent occurrence in adults. To judge from the literature it is extremely rare in patients as young as the following.

REPORT OF CASE

Rachel S., 1 year old, born in New York, was admitted to the children's ward (service of Dr. A. F. Hess) Dec. 20, 1914.

Family History.—Father and mother living and well. Two brothers and one sister alive and in good health. No history of any hereditary disease could be elicited.

Previous History.—The child was born at full term, delivery normal. Breast fed at the beginning, now receives milk three-fourths and water one-fourth; also other food from the table. No vomiting, convulsions or nervous symptoms reported. Appetite good, bowels regular.

The present illness began about four days before admission (although the child had been coughing for the past four weeks) with convulsions, dyspnea and cyanosis lasting about fifteen minutes. These symptoms subsided after an enema had been given. The child had another convulsion similar to the first, several hours subsequently, again two days ago and on the morning of day when sent to the hospital. The patient was constipated; a laxative was given, resulting in foul smelling greenish stools. There was no vomiting. Occasional cough was noticed; there was some dyspnea. Fever was marked. Since the onset of the trouble the child had been drowsy and very irritable.

On admission temperature was 101, pulse 146, respiration 40. General condition: Patient fairly well developed and nourished, slight dyspnea and cyanosis noted, no prostration or edema observed.

Eyes: Pupils equal, regular and react to light and accommodation. Petechiae present in conjunctival mucous membranes.

Nose, ears and mastoids presented no abnormalities.

Tongue clear and moist; pharynx and tonsils congested. No rigidity of neck; nasal, throat and vaginal smears negative.

In the lungs, with the exception of harsh respiratory murmur in right infraclavicular space and right axilla, the signs were negative.

The heart was not enlarged; a soft blowing murmur transmitted to the left and heard in the axilla was evident on examination. The second pulmonic was not accentuated.

The pulse was rapid, without any irregularity or intermission.

The abdomen was somewhat tympanitic, no tenderness or rigidity present. Liver was not palpable. Spleen was enlarged and could be readily felt about one finger below costal margin. The skin was hot and dry; glands not palpable, no rash present on the integument. Knee jerks active, no Babinski present.

BLOOD EXAMINATIONS

Date	12/21/14	12/23/14	12/27/14	1/2/15
Number	4,2000,000	.....	.....	3,100,000
Hemoglobin	75%	.....	.....	65%
White Blood Cells	27,000	24,000	20,000	26,000
Small Mononuclear	48%	.....	.....	.....
Large Mononuclear	6%	27%	36%	30%
Transitional	1%	1%	.....	.....
Polynuclear	45%	72%	64%	70%

Urine: Dec. 22, 1914: Straw color, acid, faint trace albumin, no sugar, acetone or diacetic acid — no bile, blood or pus. Same result when examined three days later.

Roentgenoscopy of joints and chest negative.

Dec. 28, 1914: Ears examined by Dr. Wolff Freudenthal; both drums normal.

Dec. 29, 1914: Blood culture revealed *Streptococcus attenuans.*

Bacteriologic Report by Dr. J. J. Hertz, Bacteriologist to Beth Israel Hospital.—Five c.c. of blood was obtained from the vein at the elbow, and divided in equal parts into two flasks, one containing 100 c.c. of 2 per cent. glucose bouillon and the other 100 plain bouillon. At the end of twenty-four hours the plain bouillon showed no growth. The glucose bouillon showed a streptococcic growth in chains of two to seven cocci. The organism was plated on blood agar plates and the colonies showed hemolysis. At the end of five days, the plain bouillon showed no growth.

January 1 with the change in the service, the patient was transferred to me. I take this opportunity to extend thanks for the courtesy of the case to Dr. Hess, attending physician.

Child well developed and nourished, was pallid, extremely irritable and restless, so that a thorough examination of lungs was not feasible. A distinct systolic murmur propagated to the left, was heard over the precordial region. Heart slightly enlarged. impulse fair. Spleen distinctly to be felt. Liver apparently normal; no jaundice present. Eyes somewhat sunken with dark circles underneath, face dirty waxy color. Tenderness over lower part of sternum, on which E. Libman lays stress, could not be elicited because of apathetic condition of patient.

This morning the child developed large patches of hemorrhagic spots about 2 cm. in diameter, over both cheeks and upper part of left chest. In addition small spots over right side of forehead. The rapid appearance of the patches on the cheeks led the parents to imagine that the child had been slapped and abused. It was with the greatest difficulty that the father was convinced of the falsity of any such assumption.

Jan. 2, 1915: Punctate hemorrhages under left eyelid noticed this forenoon. Weakness progressive. Color more dusky; apathy more pronounced.

Jan. 3, 1915: Bloody stools and vomitus to-day; child failing.

Jan. 4, 1915: Progressively growing weaker; respiration more rapid; pulse rapid and irregular; color more sallow, anemia more marked; died of cardiac failure about 6 a. m.

Necropsy Report by Dr. Eli Morchowitz, Pathologist.—Lungs: Do not collapse. Pleura covered by numerous petechiae. Both lungs feel solid, only portions of upper lung are crepitant. On section lungs are salmon pink in color, firm, consolidated and fleshy in appearance. Bronchial nodes are not enlarged. Slight edema of both lungs.

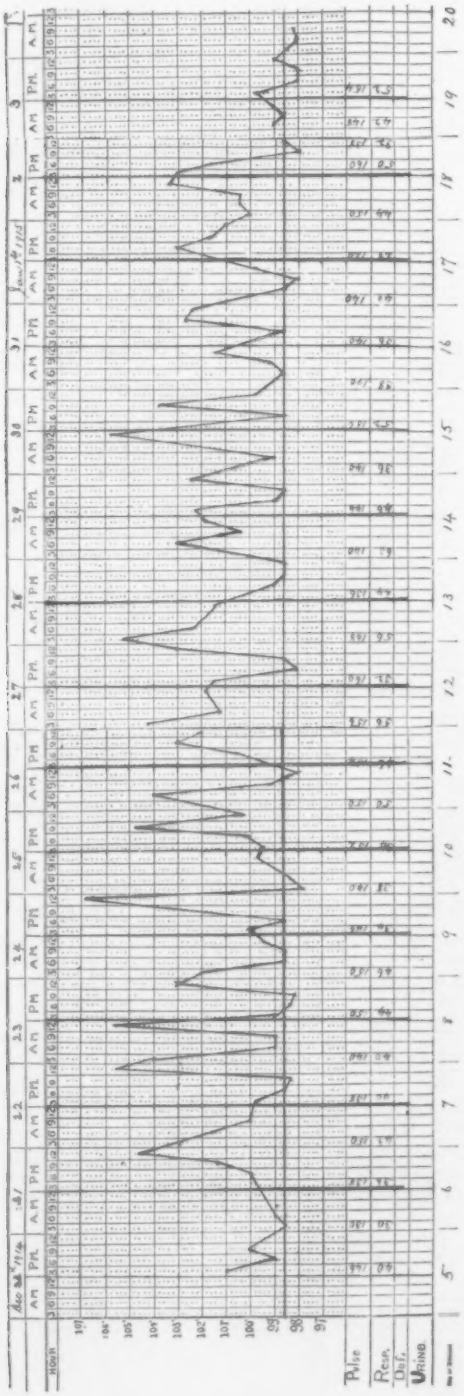

Fig. 1.—Temperature chart.

Heart: Slightly enlarged. Left ventricle moderately dilated and slightly hypertrophied. The auricular surfaces of mitral flaps are covered by numerous firm verrucous-like vegetations, the largest projections being close to edges of valves. These vegetations are more extensive on the posterior flap where they extend upward on the auricular wall almost to opening of pulmonary vein. The vegetations are in part covered by a thin layer of fresh fibrin. In the neighborhood of the vegetations are a number of ecchymoses on the endocardium. The other valves of the heart are normal. Muscle slightly flabby and pale red.

Liver: Greatly enlarged; surface smooth. On section it is intensely cloudy, markings being very much obscured.

Gall Bladder: Contains green bile. Otherwise normal.

Spleen: Slightly enlarged, capsule tense, surface smooth and firm. On section, the pulp was firm and purplish red, malpighian bodies distinct, trabeculae prominent. Pulp does not scrape. There are two small fresh white infarcts just beneath the capsule.

Kidneys: Normal in size. On section very cloudy, markings indistinct, bases of pyramids congested. Scattered over the surfaces of both kidneys are a few minute petechiae surrounded by a narrow pale zone which extends for a short distance into cortex. Capsule not adherent.

Adrenals: Normal.

Intestines: Normal.

Mesenteric Lymph Nodes: Not enlarged.

Anatomic Diagnosis: Acute verrucous endocarditis of mitral valves; slight edema of both lungs; pneumonia of heart disease (bilateral); hypertrophy and dilatation of left ventricle; cloudy swelling of heart muscle; cloudy swelling of liver; chronic congestion of spleen with fresh infarction; cloudy swelling and chronic congestion of kidneys with fresh infarctions.

Microscopic Findings: Lungs: Walls of alveoli are slightly thickened; the capillaries are dilated. The majority of the alveoli are either partially or completely filled by red blood cells, fibrin and large cells of endothelial type containing a small amount of pigment. The bronchi and pleurae are normal.

Heart: The striae of heart muscle cells are obscure, nuclei stain faintly. Otherwise normal. Heart valve mitral is covered by masses of necrosed fibrin in which are numerous clumps of bacteria. The entire valve has been converted into granulation tissue. There is an abundant round cell and polynuclear infiltration and formation of new blood vessels. The muscular wall of the heart adjacent to the valve is infiltrated with young connective tissue and formation of young blood vessels. This tissue separates the muscular bundles from one another.

Liver: Liver cells stain faintly, appear very granular, nuclei are pale. The liver cells contain a large number of fat globules. The capillaries around central veins are slightly dilated and the liver trabeculae in these regions are correspondingly narrower, the cells showing more pronounced granular and fatty degeneration.

Stomach: Normal.

Spleen: The splenic veins, especially in the cortex, are greatly dilated. The pulp is considerably congested. The center portions of the malpighian bodies stain lightly and consist of a delicate stroma of fine fibrillary connective tissue lined by large spindle cells of endothelial type.

Kidney: The epithelial cells of the tubules are coarsely granular; the cell outlines are indistinct, nuclei are sharply outlined. Most of the tubules contain a greater or less amount of granular material. The glomeruli are engorged with blood and the capillaries between the tubules are dilated and the capsule of Bowman reveal no changes. The infarcts are represented by a wedge shaped necrotic area in which the outlines of the tubules are distinctly visible. The

infarct is surrounded by a well defined narrow zone of young connective tissue proliferation. The lymph nodes of mesentery are slightly edematous.

Uterus: Normal.

The treatment is symptomatic and unsatisfactory. Antistreptococcic serum has proved efficacious in a few cases. Brodbent thinks the vaccine treatment affords some promise of success. Dr. R. Abrahams[3] reports the case of a girl 22 years of age, who recovered, after three injections of auto serum.

NOTE.—Since the above was reported, another case in a girl about 4 years of age came under observation in the Children's Service of Beth Israel Hospital. The patient was admitted for rheumatic endocarditis and on routine examination of the blood streptococcus alternans was found. The case will be reported in detail at a later date.

209 East Seventeenth Street.

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3. Abrahams, R.: New York Med. Jour., Dec. 19, 1914.

CATARRHAL JAUNDICE IN INFANCY*

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Catarrhal jaundice, or acute duodenitis with icterus, is a common disease in children between the ages of 3 and 6 years. It is, however, only infrequently met in children under 2 years of age, and is extremely rare during the first year of life. Holt,[1] for example, states that he has never seen a case in a child under 2 years of age. Henoch,[2] the "father of pediatrics," saw only two cases in children under 1 year in all his wide experience, one of these being 8 weeks, the other 5 months old. Flesch,[3] writing from Bókay's clinic, classes catarrhal jaundice in infants under 1 year as among the greatest rarities, and in looking over the records of Bókay's "Stefanie Children's Hospital" for a period of ten years, found only one typical case (a baby 1 month old) among 160,000 admissions. Starck[4] reports 65 cases of icterus in children with none under one year. L. Nicolaysen[5] (Christiania) reports 49 cases of icterus in children, not one of whom was under 2 years of age. Skormin[6] (writing from Heubner's clinic) reports three cases, one of which occurred in a month old babe, one in an infant 14 days old, and the third in an infant only 4 days old. The last two children died, the former showing no hepatitis postmortem, but colon bacilli and a coccus in the liver and blood, and the latter (the 4 day infant) showing a duodenitis with hemorrhage into the brain and meninges. These appear to be doubtful cases at best. Neumann[7] reports 430 cases of catarrhal icterus in children, with 6 in the first year of life, and 70 in the second and third years together. Langer[8] reports 144 cases in children, with one in the first year, and 19 in the second and third together.

In view of the rarity of this condition, it seemed worth while to place the following three cases on record.

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* From the Department of Pediatrics, Washington University, and the St. Louis Children's Hospital; read before the St. Louis Pediatric Society, March 19, 1915.

1. Holt, L. E.: Diseases of Infancy and Childhood.
2. Henoch: Lehrbuch der Kinderheilkunde, Ed. 9, p. 571.
3. Flesch, H.: Jahrb. f. Kinderh., 1904, lx, 776.
4. Starck: Jahrb. f. Kinderh., 1898, xlvii, 215.
5. Nicolaysen, L.: Deutsch. med. Wchnschr., 1904, No. 24, p. 878.
6. Skormin, B.: Jahrb. f. Kinderh., 1902, lvi, 176.
7. Neumann, H.: Deutsch. med. Wchnschr., 1899, No. 35, p. 574.
8. Langer: Prag. med. Wchnschr., 1905, No. 23.

CASE 1.—Baby John S., aged 8 months. Father and mother living and well. First child. No miscarriages or history of lues in family. Bottle fed baby (cow's milk formula) because of insufficient breast milk. Baby under observation from birth, has never shown any signs of lues, and, with the exception of a period of fat indigestion several months earlier, has always gained normally. Became ill Oct. 19, 1914, with increasing anorexia and drowsiness, which did not become alarming enough to suggest calling a physician until three days later. October 22, baby developed a temperature of 104, loose bowels, and increased somnolency. The next day the baby was distinctly jaundiced, and had six white, pasty, foul-smelling stools, with here and there small, black, tarry masses which gave a positive guaiac blood test. The urine showed bile. The white, pasty stools, from six to ten in number, persisted for three days, during which time the baby was intensely jaundiced and had fever up to 102-104 (rectal). The liver was enlarged and tender, and the spleen was palpable. Schmidt's test for bilirubin and urobilin in the feces was negative. October 26, the baby's temperature was normal; he was less jaundiced, and the feces were a light yellow in color. From this time on he made a rapid recovery, so that October 28, six days after the onset of severe symptoms, and nine days after the first sign of being ill, he was apparently well again. The spleen returned to normal with the drop in temperature, but the liver remained somewhat enlarged for several weeks longer. The only treatment was the substitution of skimmed milk for the whole milk formula.

CASE 2.—Baby Max S., aged 11½ months, was referred to the St. Louis Children's Hospital because of vomiting and diarrhea. Family history negative. Father and mother living and well, no history of lues or tuberculosis, the patient the youngest of a large family. On admission liver and spleen were not palpable, urine was negative, and von Pirquet and Wassermann were negative. Nov. 2, 1914, several weeks after admission, while the baby was apparently doing well, he developed jaundice, the skin and sclerae became yellow, and the urine showed bile. The stools became white and pasty. The liver was four fingers' breadth below the costal margin, and tender. November 1 he had four light yellow stools; November 2, the day on which jaundice was first noticed, he had three white pasty stools, November 3, one white stool, November 4, one light yellow stool, and thereafter a gradual return to normally colored feces. The temperature during this period ranged from 101 to 103, and there was distinct drowsiness. The jaundice cleared up as the stools regained their normal color, but he continued to run an irregular temperature. Three days later (November 6) the baby developed a cervical adenitis which went on to suppuration. November 13, the glands were incised, pus evacuated, and the temperature dropped to normal, where it remained.

CASE 3.—Baby Mary G., aged 2 months, admitted to the St. Louis Children's Hospital, June 2, 1914, with the complaint that she had "turned yellow" the week before. Father and mother living and well, no history of tuberculosis or miscarriages. The baby was the first child, and was born prematurely at 7 months, the birth weight being unknown. Never breast fed, but given condensed milk from birth. Had an otitis media at one month of age. Had a gonorrheal vaginitis on admission. One week before admission mother noticed that the baby was yellow, and this increased slightly down to the time of admission. Skin and sclerae were distinctly jaundiced. Urine was dark brown and contained bile. Mother said stools had been hard, white and constipated during the past week, but on admission were light yellow, watery, and two a day. Had had fever all week, the temperature before admission going to 104. No vomiting. When first ill, was put on a cow's milk formula. The heart and lungs were negative, the liver extended 3 cm. below the costal margin in the right mammary line, and the spleen was barely palpable. A week later, the jaundice had started to clear up; there was no more fever, the baby had gained slightly in weight, and was passing normal stools. From this time on the jaun-

dice rapidly cleared up and the liver became normal in size. This baby gave a positive Wassermann, but the course of the jaundice was typical of the catarrhal variety and there is little doubt but that it should be classed as such.

Just why this condition should be so rare in infancy seems hard to understand, for it is precisely at this age that children are most subject to gastro-enteric disorders, either through errors of diet, or through bacterial infection, two factors which are supposed to favor the occurrence of catarrhal jaundice. Perhaps, as Finkelstein[9] and others suggest, the explanation lies in the peculiarities of the infant diet; milk, as modified by the digestive juices, may become unsuitable for the multiplication of the specific bacteria or toxin necessary to produce the symptom complex under consideration. Until we know more of the etiology, however, this question can be answered only in theory.

Catarrhal jaundice in infancy differs very little from the familiar picture seen in older children. The causative factor is generally considered to be some infection, and indeed, epidemics of jaundice have been described by numerous observers. It may occur as a complication or sequel of one of the other infectious diseases, or, oftener, as a primary disease. There is usually a gastroduodenitis of varying degree, with swelling and obstruction of the common bile duct.

The attack usually begins with anorexia, coated tongue, abdominal pain of varying degree, vomiting, somnolency, slight prostration and fever. In infancy, the somnolency may be a marked feature, and there may or may not be diarrhea. Usually in two or three days, the characteristic jaundice of sclerae and skin makes its appearance, and the stools become gray or whitish, and greasy looking from the presence of unabsorbed fat. The stools are often quite foul in odor, and examination shows many fatty acid crystals. The urine is dark and shows the presence of bile. The liver is usually swollen and tender, and the spleen often enlarged. The pulse is usually slowed, sometimes markedly so, and may be irregular, but these features are ordinarily not prominent in infancy. Usually, after a few days the fever drops, the child feels better, and the jaundice disappears, but in the obstinate cases, jaundice may persist for several weeks. The liver ordinarily remains somewhat large for many days, or even weeks, after the jaundice has disappeared. The average duration of the acute symptoms, however, is from several days to two or three weeks.

The *prognosis* is almost invariably good.

The *treatment* consists mainly in dietetic measures, restricting the fats especially, and, to a lesser extent, the carbohydrates. Laxatives may be given, the salines being just as effective as the time-honored calomel. As improvement takes place, the diet may be gradually brought back to the normal.

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9. Finkelstein, H.: Lehrbuch d. Säuglingskrankheiten, Part III, p. 441.

PROGRESS IN PEDIATRICS

REVIEW OF THE LITERATURE ON THE FEEDING AND GASTROINTESTINAL DISEASES OF INFANTS FOR THE YEAR 1914-1915 *

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BREAST FEEDING

1. Von Torday: Jarb. f. Kinderh. (ref.), 1914, lxxix, 373.
2. Pritchard: Am. Med., 1914, xx, 327.
3. Misch: Ztschr. f. Säuglingsschutz, 1914, vi, 56.
4. Bahrdt: Ztschr. f. Kinderh., 1914, x, 129.
5. Brodsky: Arch. f. Kinderh., 1914, lxiii, 161.
6. Craige: Jour. Am. Med. Assn., 1915, lxiv, 502.
7. Rietschel: Ztschr. f. Geburtsh. u. Gynäk., 1914, lxxv, 732.
8. Jaschke: Ztschr. f. Geburtsh. u. Gynäk., 1914, lxxv, 736.
9. Leo-Wolf: Arch. Pediat., 1914, xxxi, 363.
10. Deresse: Ztschr. f. Kinderh. (ref.), 1914, viii, 508.
11. Hill and Simpson: Proc. Soc. Exper. Biol. and Med., 1914, xi, 82.
12. Hill and Simpson: Am. Jour. Physiol., 1914, xxxv, 361.
13. Editorial: Jour. Am. Med. Assn., 1914,, lxiii, 2292.
14. Myers: Jour. Am. Med. Assn., 1914, lxiii, 1179.
15. Van der Bogert: Arch. Pediat., 1915, xxxii, 35.
16. Neff: Jour. Am. Med. Assn., 1914, lxiii, 1181.
17. Duke: Publication U. S. Children's Bureau, No. 9, Infant Mortality Series No. 3.
18. Hirsch: Monatschr. f. Geburtsh. u. Gynäk., 1914, xxxix, 64.
19. Lateiner-Mayerhofer and Progulski: Oester., Sanitätsw., 1914, xxvi, 1.
20. Rohmer: Reichmed. Anzeiger, 1914, xxxix, 225.
21. Churchill: Jour. Am. Med. Assn., 1914, lxiii, 1799.

As in previous years, much has been written about breast feeding. While many authors have shown again by statistics the relation of breast feeding to a low infant death rate others consider this fact as well established, and are chiefly concerned with the practical difficulties presented by many cases of breast feeding.

Technic

Von Torday[1] and Pritchard[2] emphasize the fact that the regular mechanical stimulation to the breast, caused by the sucking of the infant, is very important for a plentiful milk-supply. A regular routine in lactation is considered very important by Pritchard for establishing an automatism in milk secretion; nevertheless he believes that

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the longer interval at night should be used. He emphasizes the importance of the nervous element in milk secretion; and he believes the fact that the higher nervous centers have so undoubted an influence on milk secretion can be utilized therapeutically.

Misch[3] has found that it is impossible to maintain an abundant milk-supply for any length of time when the stimulation to the breasts is insufficient, as in cases in which the mother nurses her infant only three times daily. With four feedings daily better results are obtained.

Bahrdt[4] measured the amount of suction exerted by a nursing infant, with the object of discovering the reason why some infants nurse badly. He found that the suction necessary to obtain a flow of milk varied widely; that the normal infant could suit the suction exerted to what was necessary; and that infants who nursed badly exerted less pressure than was necessary. These children showed a failure to adapt the pressure to the breast.

Brodsky[5] has studied the amount of milk secreted daily by seventeen wet-nurses in whom the breasts were completely emptied after nursing. This method he recommends as tending to maintain the milk supply. The proportion of the milk removed to that nursed was 3.4 per cent. to 46 per cent. The average daily amount secreted by the wet nurses varied from 577 gm. for 77 days to 3,500 gm. for 339 days. The highest daily quantity was 5,400 gm.

The intervals between feedings generally recommended are three or four hours. Craige[6] recommends a three hour interval, with one night feeding until the third or fourth month, after which time all night feedings should be discontinued. Some babies, he believes, do better on four hour intervals. Overfeeding due to too frequent feeding he finds very common among breast-fed babies.

In feeding new-born infants, Rietschel[7] believes that the intervals should be shorter; he warns against the systematic method so often advised of feeding new-born infants only five times in twenty-four hours, for he finds that many of these children thrive better on six to eight feedings in twenty-four hours.

Jaschke[8] disagrees with this view, and advises even in new-born infants five feedings in the twenty-four hours. If the amount of milk taken is insufficient, he advises that the breasts should be pumped, and that this milk be given after each nursing.

Leo-Wolf[9] also recommends less frequent intervals of feeding for the new-born infant. On the first day the baby should not be nursed at all, and on the second day once or twice, while water ad libitum is given. The number of feedings is increased on the third day to three, on the fourth to four; and only on the fifth day is the infant to be nursed every four hours during the day.

Deresse[10] does not find insufficient amount of breast-milk to be the principal cause of inability of mothers to nurse their infants. Among one hundred mothers, in only twenty was the cause either insufficient amount of milk or disease.

All writers seem agreed that galactogogues have no influence in increasing the supply of breast milk. Hill and Simpson[11] have investigated the effect of the injection of the extract of hypophysis on the milk secretion of cows. This has been found by some writers to have a very marked effect. The authors found that there was a decided increase in milk secretion, with a rise in the percentage of fat. This was very temporary, and was present only in the first few hours after the injection; later the changes were counterbalanced, so that the examination of the daily amount showed no changes from the normal. They[12, 13] have lately made the experiment on the human subject, injecting 20 mg. of the commercial preparation intramuscularly. This was followed in ten minutes by a marked increase in the amount of milk secreted. The fat was also increased, being 5.5 per cent. as compared with 3.4 per cent.

Nutritional disturbances on breast-feeding are considered by Myers[14] as not infrequently due to faults in the mother's diet. In this he disagrees with many recent writers, among them Van der Bogert,[15] who believes that any food, whether or not it contains acid, may be allowed unless it causes indigestion in the mother. Myers reports three cases illustrating his theory, where restlessness, diarrhea, or colic in the infant disappeared when fruits and vegetables were omitted from the diet of the mother. The food giving most disturbance he finds to be those containing the largest amount of high flavors; among these he includes fruits, spices and highly flavored vegetables. Fruit both cooked and uncooked has proved injurious. He has noticed a great variation in the sensitiveness to these substances as displayed by different babies, or by the same baby at different ages. He advises that from the beginning of lactation until the end of the first month the use by the mother of the foods named should be limited; and that after this time the addition of vegetables and fruit to the diet should be made very cautiously, only one new article being tried at a time.

Neff[16] advises that in certain disturbances in breast-fed infants, especially in those characterized by abdominal distention, colic, fretfulness, loose stools, and failure to gain, fat-free breast milk should be tried. He believes that it is also indicated in many cases with vomiting, both in those with and without pyloric obstruction, in cases of infantile eczema and of congenitally weak and premature infants. The infants are allowed to nurse a few minutes at the breast, this serving to keep

the milk secretion active. After each nursing all the remaining breast milk is expressed from the breast; this is kept on ice till the cream is separated; the skimmed breast milk is then given as supplementing each nursing.

Studies in the Distribution of Breast Feeding and Its Relation to Infant Mortality

Hirsch[18] studied the histories of the feeding of all of the children of the women visiting the ambulatorium of the Frauenklinik at Munich, and found a very low rate of breast feeding: 57 per cent. of the children were not nursed at all; only 33 per cent. were nursed longer than one month; 20 per cent. longer than three months, and 13 per cent. longer than six months. The influence on the infant death rate was shown by the fact that of those who died, only 3 per cent. were breast-fed over six months, while 74 per cent. were not nursed at all.

The statistics from the Lemberg clinic, reported by Lateiner-Mayerhofer and Progulski,[19] show a marked contrast, as here the proportion of babies breast-fed is very high. Only 10 per cent. of the infants were nursed less than six months, while 55 per cent. were nursed one year and many longer. Mixed feeding, which was often injudicious, was frequently given, almost regularly after the sixth month. The mortality among the breast-fed was 16 per cent., among the artificially fed 34 per cent. The authors call attention to the fact that though breast feeding is so general in Galicia (as illustrated by these figures) the infant death rate is very high, being over 20 per cent. This high rate they attribute to the very bad economic and hygienic conditions. They believe that hygienic instruction of the inhabitants could accomplish much.

Rohmer[20] found in Cologne that among married women from 82 to 69 per cent. nursed their babies. Of illegitimate infants, only 31 per cent. were breast-fed after the sixth week.

Among 100 French women who were unable to nurse their infants for some reason, Deresse[10] found that in twenty insufficient milk or disease was the cause, in seventy-three the social position of the mother, in seven egotism or the prejudice of the mother.

Churchill[21] has studied the relation of the feeding of breast milk to the mortality in an infant hospital for a period of three years. He found that the rates of mortality among the infants exclusively breast-fed and those fed exclusively on cow's milk were 36.3 and 36.9, respectively. This practical identity in the rate could be explained by the fact that the infants who had received exclusively breast milk were on an average younger and weighed less than those who had received cow's milk. The death rate of the babies receiving cow's milk who were of equally low weight was considerably higher than that of the infants receiving breast milk.

ARTIFICIAL FEEDING IN GENERAL

22. Pritchard: Brit. Jour. Child. Dis., 1914, xi, 49.
23. Heubner: Ztschr. f. Kinderh., 1914, xi, 81.
24. Samelson: Ztschr. f. Kinderh., 1914, xi, 86.
25. Mayerhofer and Roth: Ztschr. f. Kinderh., 1914, xi, 117.
26. Gittings: Arch. Pediat., 1914, xxxi, 696.
27. Chapin: Jour. Am. Med. Assn., 1914, lxiii, 1177.
28. Mendel: Jour. Am. Med. Assn., 1914, lxiii, 819.
29. Osborne and Mendel: Jour. Biol. Chem., 1914, xvii, 325.
30. Osborne and Mendel: Jour. Biol. Chem., 1914, xvii, 401.
31. Editorial: Jour. Am. Med. Assn., 1914, lxiii, 247.
32. Niemann: Jahrb. f. Kinderh., 1914, lxxix, 274.
33. Mendel: Biochem. Bull., 1914, iii, 156.
34. Osborne and Mendel: Jour. Biol. Chem., 1914, xvi, 423.
35. Funk: Ergebn. d. Physiol., 1913, xiii, 124.
36. Funk: Die Vitamine, Wiesbaden, 1914.
37. Funk: München. med. Wchnschr., 1913, lx, 2614.
38. Schaumann: Arch. f. Schiffs.- u. Tropenhyg., 1914, xviii, 125; Ztschr. f. Kinderh. (ref.), 1914, viii, 115.
39. Wheeler and Biester: Am. Jour. Dis. Child., 1914, vii, 169; Wheeler: Am. Jour. Dis. Child., 1915, ix, 300.
40. Peiser: Berl. klin. Wchnschr., 1914, li, 1165.
41. Smith: Arch. Pediat., 1914, xxxi, 784; LeWald: Am. Jour. Dis. Child., 1915, ix, 1261.
42. Kerr: New York Med. Jour., 1915, ci, 296.
43. Variot: Ztschr. f. Kinderh. (ref.), 1914, viii, 281.
44. Müller and Schloss: Med. Klin., 1914, x, 276.
45. Müller and Schloss: Jahrb. f. Kinderh., 1914, lxxx, 42.
46. Leopold: Am. Jour. Dis. Child., 1914, viii, 196.
47. Friedenthal: Berl. klin. Wchnschr., 1914, li, 727,

For the determination of the proper amount of food to be given, the caloric method is again advised by many authors. Several of these draw attention to Howland's work which showed that the caloric needs of infants making active movements and crying is greater than those of infants at rest. Pritchard[22] believes that infants who are overheated by too warm rooms or too thick clothing require less food than infants properly cared for.

Heubner[23] states that to obtain in the artificially fed infant a rate of gain equal to that in the breast-fed a higher number of calories is required. Samelson[24] does not agree with this conclusion.

Mayerhofer and Roth,[25] basing their statements on observations of twenty-three infants so fed that they show very good weight curves, conclude that a perfect uniformity in the caloric needs of infants does not exist. There are many infants who gain best when the calories given fall below the standard of 100 calories per kilogram of body-weight recommended by Heubner; other infants thrive only on an energy quotient far above 100. Heubner's standard, they consider, is the best foundation for the comparison of different forms of food.

The fact that the caloric needs vary with different infants is also emphasized by Gittings;[26] he finds that atrophic children especially

require a higher energy quotient than that corresponding to their weight. He believes that a caloric standard is a useful check in artificial feeding; for if the caloric value is much above or below the average, harm will result in the majority of cases. In Chapin's[27] opinion, the experiments on animals showing the different results obtained with different foods which yield the same number of calories should be a warning not to ascribe too much importance to the caloric method.

The interesting experimental studies made this year as to the influence of different food elements on nutrition and growth, and on the disturbances produced by the absence of certain necessary substances or vitamines in food, have been frequently commented upon in pediatric literature.

Chapin[27] believes that in infant feeding the facts brought out by experimental studies in nutrition should be borne in mind; that a food may be very unscientific (even though exactly prepared with some supposed chemical or caloric requirement in mind), in case some necessary element has been omitted. He warns against the intricate and profound chemical manipulation of food, and believes that the latest scientific studies point favorably to great simplicity in the preparation of artificial food for infants. In feeding he believes that cow's milk should be simply diluted in the right proportion for the age, keeping in mind the fact that none of the ingredients, protein, fat or carbohydrate must be long reduced below what is known to be their average content in human milk.

The important studies of Osborne and Mendel[28, 29] have thrown much light on the problems of the nutrition of the growing infant. Of the amino-acids into which the ingested protein is broken down during digestion and metabolism, almost all are necessary for growth; only certain ones are, however, necessary for maintenance of life. Tryptophan is one of the latter. Zein, contained in maize, does not contain tryptophan, and does not maintain life in animals if fed as the only protein. If tryptophan is added to the food, the animals will live. Lysin has been found by these authors to be essential for growth. Gliadin, which is deficient in lysin, maintains life in adult animals, but will not cause growth in young animals. If lysin is added to this diet the animals grow. Zein, which lacks tryptophan, is also lacking in lysin; Mendel concludes that this fact explains why corn is poorly suited to the growing individual. Proteins like casein, lactalbumin and egg vitellin, which in nature are concerned with growth, are rich in lysin; these, with edestin from hemp seed, are capable of satisfying the nitrogenous needs of all states of development. For growth certain fats have been found by these investigators[28, 30] to be essential. Growing animals fed on isolated food substances with fat in the form of

lard, stopped growing; growth, however, was restored if butter fat was added. Butter fat, egg yolk and cod liver oil contain the ingredient which promotes growth; almond oil and lard do not. The former foods, they remark, have long had the reputation of being efficient foods. They believe that it is improbable that their special potency resides in the triglycerids present. Their work has been commented on[31] as throwing light on the experiments of Niemann[32] in adding butter fat to the diet of infants.

The tendency to grow is, according to Mendel,[33] dependent on hereditary factors; food gives a range of play, but does not influence it permanently. Osborne and Mendel in their experiments on rats, have by various means stopped growth, without causing a permanent loss of ability to grow. They conclude that the tendency to grow apparently does not disappear until the fundamental tendency of the organism is used up.

The term "vitamine" was first used by Funk to describe certain substances present in food in small quantity but necessary to nutrition "which can not be expressed in terms of protein, fat and carbohydrate, or in calories of energy" (Mendel[28]). Mendel believes that classing these unknown substances under the name of vitamines does not explain the disturbances arising from their absence from the diet. Funk[35, 36, 37] has written much on the subject. He describes vitamines as crystalline bodies of very complicated structure containing nitrogen, which can be isolated by different precipitating agents. These bodies are necessary for life; they are thermolabile and are present in very small amounts. They are biologically if not chemically associated. Vitamines are present in the polishings of rice and in milk. Among avitaminoses or deficiency diseases, due to a lack of vitamine in the food, he classes beriberi, scurvy, Barlow's disease, pellagra and rachitis, *Mehlnährschaden* and many other syndromes.

Schaumann[38] considers that Funk's conclusions in regard to all these diseases are theoretical, and have not yet been proven.

Wheeler and Biester[39] have studied experimentally certain proprietary foods used in infant feeding with the object of determining whether they will sustain life and growth when used as exclusive foods; and if not whether the fault lies in a lack of the essential amino-acids, or in the absence of essential accessory substances or vitamines.

Many authors warn against one-sided diets; especially against those very rich in carbohydrate but poor in fats which have been extensively used. Chapin[27] believes that the withdrawal of the fats brings with it especial danger of rachitis.

Niemann[32] believes that fat in the diet is especially necessary for the accumulation of body fat; and that the diets very rich in carbohydrate

tend to decrease immunity. Peiser[40] reminds us that the feeding of normal infants on diets poor in fat and rich in carbohydrate is not "economical"; and that the resulting increased water content of the body tends to lower the immunity.

Technic

Smith[41] believes the posture of the baby during feeding is very important; and advises that immediately before and after feeding the baby should be held upright in order that swallowed air may be expelled. Many infants, he finds, swallow large quantities of air with their feeding. With the baby in the recumbent position fluid gravitates to the posterior portion of the stomach covering the cardiac orifice; the air therefore cannot escape through the esophagus, and leads by pressure to vomiting and colic.

ARTIFICIAL FEEDING OF THE NORMAL INFANT

The simple milk dilutions, so widely used, are very little discussed in the literature.

Kerr[42] favors top-milk mixtures. Variot[43] gives during the first five or six weeks one part of water to two of milk; in the third month one part of water to three of milk; at the fourth to the fifth month milk is given undiluted. Müller and Schloss[44] give directions for the preparation in the private house of the most important of the milk mixtures.

Two foods were described in this review last year which had been suggested as being superior to the simple milk dilutions in feeding the normal infant: Friedenthal's milk and the whey-modified milk of Schloss. Both have been developed on the principle of imitating breast milk as nearly as possible. Müller and Schloss[45] criticize the article by Bahrdt on Friedenthal milk, on the grounds that according to Bahrdt's analyses the salts in this milk do not resemble, as they are supposed to do, those of breast milk either in total content or in the relative proportions of the various salts; and that the clinical results reported are inconclusive because there were but few young infants fed on the mixture, and because the duration of the feeding was too short. They describe the stages in the development of the "whey-modified milk" of Schloss. They have found that a simple dilution of milk and cream with the addition of potassium chlorid forms a food very similar in composition to that of human milk, both in absolute salt content, and in the relationship of the various ions. They have found that milk-sugar, which they added at first, often leads to bad results, especially in infants' hospitals; they have therefore added dextrin and maltose as the carbohydrate. They discuss the various attempts which have been made during the development of infant feeding to produce an artificial food similar to breast milk.

Leopold[46] has described his results in the use of the whey-modified milk of Schloss. The preparation of the two mixtures prescribed by Schloss are once more described.

Mixture A, for infants under three months, consists of:

Twenty per cent. cream	140 c.c.
Whole milk	140 c.c.
Water	700 c.c.
Potassium chlorid	0.2 gm.
Dextrin and maltose	35.0 gm.
Flour	15.0 gm.
Nutrose or plasmon	5.0 gm.

In Mixture B, for infants over three months: the flour is omitted, and the dextrin and maltose is increased to 50 to 70 gm.; otherwise mixtures A and B are the same. During the past year Leopold has given this feeding to fifty-four infants, varying in age from 2 days to 13 months. Sixteen were under 1 month. Two died; one from double otitis media, one from peritonitis. The milk was given in the same amounts as breast milk. The stools were alkaline, yellow, homogeneous and pasty. Leopold has found that infants over 6 months do not gain well on this feeding. He considers that the food is especially indicated for very young infants for whom breast milk is not obtainable. Of the fifty-four cases, in thirty-six the result was good; in ten fairly good, in eight poor. He concludes that the results with this feeding are much better than with any other method of artificial feeding.

Friedenthal[47] writes that in foods for infants the correlation of salts is more important than the total ash content of the milk. The low salt content of human milk is the optimum for the normal child.

48. Lichtenstein: Hygiea, 1914, lxxvi, 17; Monatschr. f. Kinderh. (ref.), 1914, xiv, 7.
49. Modigliani: Pediat., 1914, xxii, 405.
50. Di Cristina: Pediat., 1914, xxii, 161.
51. Langstein: Jahreskurs. f. ärztl. Fortbild., 1914, v, 54.
52. Sluka and Sperk: Wien. klin. Wchnschr., 1914, xxvii, 833.
53. Aschenheim: Therap. Monatsh., 1914, xxviii, 435.
54. Kern and Müller: Berl. klin. Wchnschr., 1913, l, 2237.
55. Soldin: Berl. klin. Wchnschr., 1914, li, 794.
56. Leichtentritt: Arch. f. Kinderh., 1914, lxiii, 61.
57. Rost: Arch. Pediat., 1914, xxxi, 849.
58. Beck: Med. Klin. 1914, x, 149; Ztschr. f. Kinderh. (ref.), 1914, viii, 129.
59. Lust: Ztschr. f. Kinderh. (ref.), 1914, viii, 129.
60. Moro: Ztschr. f. Kinderh. (ref.), 1914, viii, 129.
61. Ostrowski: Jahrb. f. Kinderh. (ref.), 1914, lxxix, 374.
62. Wegener: München. med. Wchnschr., 1914, lxi, 359.
63. Kamnitzer: Deutsch. med. Wchnschr., 1914, xl, 855.
64. Bosworth and Bowditch: Am. Jour. Dis. Child., 1914, viii, 120.
65. Miller: Arch. Pediat., 1914, xxxi, 772.
66. Eaton: Arch. Pediat., 1914, xxxi, 452.
67. Dunn: Amer. Jour. Dis. Child., 1915, ix, 225.
68. Dunn: Boston Med. and Surg. Jour., 1915, clxxii, 167.

Albumin Milk

The value of this food and its substitutes in the feeding of sick infants is once more attested by many authors. Lichtenstein[48] reports fifty cases, for the most part cases of severe nutritional disturbance, treated with albumin milk. The results were in the main very good. He found it possible usually to change to another diet after two to three weeks.

Modigliani[49] reports good results with albumin milk in the feeding of six infants with dyspepsia, intoxication and balance disturbance, as shown by a gain in weight, cessation of fermented stools, and the appearance of fat-soap stools. Two breast-fed infants with dyspepsia recovered when albumin milk was fed as a supplementary food. The results when albumin milk was used in the feeding of eight new-born infants were very bad.

Di Cristina[50] has had more favorable results in feeding albumin milk to normal infants. He used it also in all cases of nutritional disturbance. In chronic dyspepsia he has seen a prompt improvement in all the symptoms; in these cases where the weight remained stationary fat and sugar were added.

Langstein[51] writes that for severe cases of nutritional disturbance albumin milk is superior to casein calcium milk. Sluka and Sperk[52] found that feeding with albumin milk was more successful with dispensary cases than with hospital infants. The poor results with the latter they ascribe to "hospitalism."

Substitutes for Albumin Milk

In all the preparations the object has been to obtain a mixture with a low salt and sugar content, and a high percentage of protein and calcium.

With regard to the many substitutes for albumin-milk, Finkelstein (see Footnote 137) writes that he prefers those which contain casein in fine, freshly precipitated flakes. The powdered casein preparations, such as casein calcium, plasmon, etc., may become hard and tough.

Aschenheim[53] has suggested a new modification of albumin milk.

Kern and Müller[54] have also proposed a new substitute. One liter each of buttermilk and water are boiled together and the amount filled up to two liters. The mixture on standing shows a precipitation of the casein, and a formation of clear whey; 1125 gm. of the whey are removed, and 125 gm. of 20 per cent. cream are added to the remainder, together with sugar from 5 to 7 per cent. The advantages claimed for this food are its freshly precipitated casein and its content of lactic acid.

Soldin[55] has prepared a similar protein-rich food. Half a liter of milk is allowed to sour; the cream is removed, and the milk is warmed

to 40 C. The casein which is precipitated is separated by a sieve, and is then added to 0.5 liter of sour milk and 0.5 liter of water or flour water with sugar. The food is prepared easily and is cheaper than albumin milk. The clinical results have been good.

Leichentritt[56] describes the preparation of Engel's modification of albumin milk, which was reviewed last year. His results with this feeding were good, as he shows by case histories.

Casein-calcium milk has been used to a considerable extent.

Rost[57] reports fifty-six dispensary cases of summer diarrhea treated with a mixture prepared according to Stoeltzner's directions. Of the casein-calcium preparation 20 gm. are added to ⅓ of a pint of milk; ⅔ of a pint of milk is heated; the cold solution is added and the whole brought to the boiling point; one pint of boiled water or cereal decoction is added. Rost rarely added sugar (in the form of dextrin-maltose) before the third day. The histories of the cases show that the results were, in the main, good. Twenty-seven showed gain in weight. Rost recommends this food on account of the simplicity of the preparation and its cheapness as particularly useful in the treatment of dispensary cases.

In acute and sub-acute dyspepsia Beck[58] has had as good results with casein calcium milk as with albumin milk; in intoxication, however, the results have been less good. Lust,[59] in discussing Beck's report, states that his own results with casein-calcium milk has been very unequal. He does not believe that this preparation replaces albumin milk. Moro,[60] too, found that in older infants and less severe cases the results were excellent; he has seen some bad effects of casein-calcium milk in other cases.

Ostrowski[71] fed with casein-calcium milk forty-nine infants with the different nutritional disturbances; his results were very good. Wegener[62] used this food successfully in twenty-two cases. Kamnitzer[63] reports eighteen cases in which casein-calcium milk was used; two very young infants with intoxication died. In the others, however, there was improvement, the stools becoming formed on the second to fifth day. Langstein[51] has noted, in slight and moderate cases of nutritional disturbances, results with casein-calcium milk equal to those of albumin milk; in severe cases, as already noted, albumin milk is superior.

In connection with these methods for increasing the proteid content of a milk mixture, the preparation of dry powdered paracasein, as described by Bosworth and Bowditch,[64] is interesting. Very large quantities of this substance, when fed to an infant, led to no toxic symptoms nor fever.

Other Diets

The diets of the group including albumin milk and its substitutes are in general recommended chiefly for those disturbances ascribed to the harmful action of too high concentrations of sugar and salt. For disturbances associated with an intolerance for fat, other diets are recommended.

For these cases Miller[65] and Eaton[66] write of the good effects of a diet rich in protein and carbohydrate, and poor in fat. Miller[65] gives at first a diet with a very low percentage of fat (0.5 per cent.) moderate protein (from 1.3 to 1.5 per cent.) and high carbohydrate (from 5 to 6 per cent.). The fat is increased very slowly, the proteid and carbohydrate more rapidly. On this diet infants show putty-like stools and make a marked improvement. Eaton[66] also reports infants doing well on a diet with very little fat. For cases of fat intolerance, both acute and chronic, Dunn[67, 68] advises a diet rich in protein (3 per cent.) with moderate carbohydrate (7 per cent.) (which should be in the form of maltose) and very low fat. For some cases he considers breast milk essential. Niemann,[52] basing his method on his theory that injury by fat is due to its content of the lower fatty acids, recommends feeding infants with fat intolerance on mixtures made up from skimmed milk and washed butter. By washing butter thoroughly, and kneading it as the wash water is renewed eight or ten times, it may be largely freed from the lower fatty acids. He has used the following diet:

500 gm. skimmed milk.
500 gm. 5 per cent. flour water (mondamin).
50 gm. malt extract.
50 gm. washed butter.

Peiser[40] has made experiments to determine which fats are best tolerated by infants. Using buttermilk with flour as the medium, he has added (usually for eight-day periods) different kinds of fats: cod liver oil, salt-free butter, 15 per cent. cream and many other oils. The impression gained from his results is that cod liver oil is tolerated the best, butter and cream the least well.

THE ETIOLOGY OF NUTRITIONAL DISTURBANCES OF INFANTS

69. Heubner, W.: Arch. f. exper. Path. u. Pharmakol., 1914, lxxv, 435.
70. Rietschel, Heidenhain and Ewers: München. med. Wchnschr., 1914, lxi, 648.
71. Lövegren: Ztschr. f. Kinderh., 1914, xii, 110.
72. Moro: Jahrb. f. Kinderh., 1914, lxxix, 645.
73. Hahn and Moro: Jahrb. f. Kinderh., 1914, lxxix, 664.
74. Hayashi: Jahrb. f. Kinderh., 1914, lxxix, 674.
75. Klocman and Moro: Jahrb. f. Kinderh., 1914, lxxix, 676.
76. Luna: Pediatria: 1914, xxii, 88.
77. Gismondi: Pediatria, 1914, No. 4; rev. Arch. Pediat., 1915, xxxii, 151.
78. Sherman and Johnes: Arch. Pediat., 1914, xxxi, 749.

79. Southworth: Jour. Am. Med. Assn., 1914, lxiii, 1375.
80. Freudenberg: Monatschr. f. Kinderh., 1914, xiii, 141.
81. Schlutz: Journal-Lancet: 1914, xxxiv, 178.
82. Brüning: Jahrb. f. Kinderh., 1914, lxxix, 305.
83. Thomas: Biochem. Ztschr., 1913, lvii, 456, 473.
84. Bahrdt and McLean: Ztschr. f. Kinderh., 1914, xi, 143.
85. Huldschinsky: Ztschr. f. Kinderh., 1913, v, 475.
86. Benjamin: Jahrb. f. Kinderh., 1914, lxxx, 545.
87. Lesné: Ztschr. f. Kinderh. (ref.), 1914, viii, 117.
88. Lust: München. med. Wchnschr., 1913, lx, 2720.
89. Lust: Jahrb. f. Kinderh., 1913, lxxvii, 383.
90. Lawalschek: Prag. med. Wchnschr., 1914, xxxix, 185.
91. Hayashi: Monatschr. f. Kinderh., 1914, xii, 749.
92. Modigliani and Benini: Policlinico, 1914, xxi, No. 51; (rev.) Jour. Am. Med. Assn., 1915, lxiv, 475.
93. Poulsen: Jahrb. f. Kinderh., 1914, lxxix, 77.
94. Uffenheimer: Jahrb. f. Kinderh., 1914, lxxx, 543.
95. Uffenheimer: Jahrb. f. Kinderh., 1914, lxxix, 92.
96. Liwschiz: Dissertation, Munich, 1913.

The two main causes of the nutritional disturbances in infancy, food and bacterial infection, have been discussed as in previous years.

The influence of the various foodstuffs in the etiology of the nutritional disturbances of infants is always a fruitful subject of discussion in pediatric literature.

Influence of Food and Foodstuffs: Salts

The whey salts in cow's milk are considered by most German writers as being extremely important in causing nutritional disturbances.

The much-mooted question of salt fever is still debated. Heubner,[70] although acknowledging the importance of the freshness and sterility of the diluting fluid in preventing a rise of temperature after the subcutaneous injection of salt solution, still believes that the results of certain experiments speak for the action of an excess of sodium chlorid in causing fever.

Rietschel, Heidenhain and Ewers[71] deny that all cases of fever arising after the subcutaneous injection of salt solution may be ascribed to bacteriotoxic substances in the distilled water used, and believe that in some cases an inorganic constituent such as copper may be the cause. In proof of this theory they report that 58 per cent. of the infants injected with freshly distilled solutions which had been boiled with bits of copper showed a rise in temperature.

Finkelstein has believed that the alimentary fever occurring in young infants and in those with nutritional disturbances after the taking by mouth of salt solution is caused by a biochemical action of the salt on the organism, and not, as thought by some authors, that this fever is due to bacterial factors, becoming active after an injury to the intestinal epithelium by the salt. Lövegren[72] believes that this question

can not be settled clinically; he has therefore subjected it to experiment. In healthy dogs, during the height of digestion, he has injected salt and sugar solutions directly into the mesenteric veins, thus excluding any action of the salt on the intestinal epithelium. In these operations the greatest care was used to maintain asepsis; the solutions used were made from freshly distilled water and then autoclaved; for most of the distillations a glass apparatus was used. Experiments were made with hypertonic, hypotonic and isotonic salt solutions, with distilled water, with lactose and dextrose solutions, and with other solutions. In eleven cases in which a hypertonic solution was injected there was ten times a rise of temperature; one case with hypotonic solution also showed a rise; while in one case each injected with an isotonic solution and with Ringer solution the reaction was weak. He concludes that in his experiments the fever must be due to a direct action of an anisotonic solution on the body cells.

It does not seem excluded that the rise in temperature (averaging 1.1 C.) may not have been due to the operative procedure; the two cases used as controls, in which an isotonic solution and Ringer's solution were injected, which showed but a slight rise in temperature, seem insufficient.

Moro[72] and his co-workers[73, 74, 75] have made an interesting series of experimental studies in regard to the action of whey salts on the intestinal epithelium.

Carbohydrate

Luna[76] writes that infants with a flatulent dyspepsia, and positive fermentation test as applied to the stools reveal an intolerance of carbohydrate. These children should be fed very little carbohydrate and much albumin.

In regard to which form of sugar is least likely to cause nutritional disturbance, Gismondi[77] believes that a healthy child is able to digest lactose, saccharose and maltose equally well. He proposes giving a mixture of the three varieties, the object being not to exceed the limit of tolerance of each kind. Sherman and Johnes[78] have tested the various sugars for their effect on the gastric secretions of fifteen infants. They conclude from their results that malt sugar in the form of dextrin maltose is at least twice as stimulating to gastric secretion as milk sugar or cane sugar.

That starch has a protective action against the injurious effects of both sugar and fat is claimed by Southworth.[79] He does not agree with the view that the beneficial results obtained by the use of starch gruels is due solely to their prevention of the formation of tough curds. He believes that there are several ways in which starch may influence digestion. The protective action of starch is shown by the benefit

derived from its use in malt-soup, which contains a high proportion of sugar. He believes that the slow breaking down of starch gives less opportunity for fermentation.

The symptom complex of *Mehlnährschaden,* due apparently to a diet consisting too exclusively of carbohydrates, has been much discussed. Funk, as previously stated, has included this disturbance among the "avitaminoses" or deficiency diseases. This view has been opposed by Schaumann, and especially by Freudenberg.[80] The latter finds fault with Funk's hypothesis, which attributes to a lack of vitamines a disease so plainly associated with a lack of many of the constituents of a sufficient food. Schlutz[81] reports a case illustrating the hypertonic form of this disease. A child 14 months old, fed for two months on a gruel without milk, developed edema, stiffness and contractures, but recovered on breast milk. Brüning[82] has contributed to our experimental knowledge of this disturbance. White rats, fed on a one-sided carbohydrate diet, showed on an analysis of the body tissues a greater amount of water and a higher relative amount of salt. He shows that these findings correspond with the clinical condition in *Mehlnährschaden.*

Niemann[32] and Peiser[40] comment on the reduction in immunity caused by a one-sided carbohydrate diet. This loss of immunity has frequently been commented on. Thomas[83] found that young pigs fed chiefly on carbohydrate showed a marked lessening of resistance to tuberculosis, while those fed chiefly on protein showed a limitation of the tuberculous process. Langstein[115] remarks on this work as being an experimental confirmation of the clinical facts recorded by him of the especially favorable course of infections, especially of tuberculosis in infants, when protein forms a large part of the diet. He warns, however, against drawing conclusions from experiments.

Fat

Two nutritional disturbances are ascribed to a high percentage of fat in infant-feeding: an acute, and a chronic disturbance.

Dunn[67, 68] describes cases showing an intolerance for fat, which he believes are caused more often originally by an overfeeding with carbohydrate than by one with fat. These cases are frequently ones of extreme malnutrition. They show their lack of tolerance for fat by the facts (1) that they do not gain in weight on diets low in fat, yet when it is added give evidence of failure to absorb it by the presence of free fat or excessive soap in the stools, and (2) that a further increase in fat in the diet leads to acute disturbances with vomiting, diarrhea, and frequently with collapse. The treatment recommended by Dunn for these cases has already been spoken of.

Niemann[32] describes the two classes of disturbance caused by fat:

acute disturbances associated with diarrhea and chronic disturbances or *Milchnährschaden*. There is some ground for believing that the reason why cow's milk fat is so much more apt than the fat of breast-milk to cause disturbances of both types is the fact that cow's milk contains more of the lower volatile fatty acids. In proof of this theory he shows that children who cannot take milk-fat with safety may be fed large amounts of oil, and that the best oils are those low in the lower, volatile fatty acids.

The relation of the volatile fatty acids to the nutritional disturbances is a very complicated one. Bahrdt and McLean[84] have studied this subject. They have shown that diet influences greatly the content of the stools of normal infants in free volatile fatty acids. In those of breast-fed infants the percentage was high; in those of artificially fed infants free volatile fatty acids are absent. In the stomach contents of normal infants on the two types of feeding the proportions are, however, reversed; in the stomach contents of infants on cow's milk there is from three to six times as much volatile fatty acid as in those of infants on breast milk. This result may probably be explained by the fact that the origin of the volatile fatty acids is quite different in stomach and in intestine. In the stomach they arise normally almost exclusively by a fermentative splitting of preformed butyrin or caproin; of this breast milk has less than cow's milk; in the intestine the acids are formed mainly by bacterial action on the sugars and also on the higher fatty acids. The authors found no free volatile fatty acids in the stools even when sugar or cream were added to the diet of artificially fed infants. These acids were found, however, to be present free in every case in the stools of artificially fed infants with acute nutritional disturbances and in such concentration that the authors believe that these acids have some pathologic action in stimulating peristalsis.

Huldschinsky[85] studied the content of volatile fatty acids in the stomach contents of infants with gastro-enteritis, or suffering from overfeeding. He reports that rarely was there an increase as compared with the stomach contents of normal children. He believes that these acids can only be looked on as injurious in cases in which the stomach is already disturbed in function.

Protein

There has been little written this year in regard to a directly harmful action of protein in the diet of infants.

Benjamin[86] found that large doses of "plasmon," a preparation of protein, caused protein fever in infants. Lesné[87] writes of acute cases of alimentary hypersensitiveness, both slight and severe, usually induced by eggs and cow's milk. The severe cases showed a

rapid pulse, fall in temperature and collapse. Lust[88] warns against the common practice of feeding egg albumin water in the treatment of acute nutritional disturbances in infants. In the course of his experiments in regard to the permeability of the intestinal tracts of infants to unchanged protein, he fed twenty normal infants the white of one-half to one egg; in a few cases that of two eggs. Sixteen of the children showed some gastro-intestinal symptoms; some showed frequent thin stools. Three infants showed edema and urticaria.

Lust's[89] work was reviewed last year. To discover whether or not protein passes through the intestinal epithelium unchanged he used the following test: The urine and in some case the blood serum of the infants fed on certain proteins were tested with a serum having a strong precipitation titer for the protein which had been fed. He found that in cases of acute and chronic nutritional disturbances in infants, egg albumin frequently passed through the intestinal wall unchanged.

Lawatschek[90] has had similar results, using the same tests. Among twenty-nine older infants tested, in cases of slight nutritional disturbance, he found no passage of unchanged protein; in severe disturbances, he obtained markedly positive reactions. He believes that there is a parallelism between the degree of the reaction and the outcome of the disease, and believes that the test may be used to determine the prognosis. Lawatschek, like Ganghofner and Langer previously, found that new-born infants up to the tenth day constantly gave evidence of the passage of unchanged foreign protein through the intestinal wall. All of the new-born infants who did not vomit the egg albumin gave positive reactions.

Hayashi[91] has attempted to determine the limit of tolerance of the intestine of twenty-eight normal infants for egg albumin, using the same technic as Lust. He has found that when from 15 to 20 gm. egg albumin are given to the normal infant, the unchanged proteid can usually be demonstrated in the urine. This limit of tolerance he has not found to undergo much change during the first year. In infants with exudative diathesis and in infants who have recovered recently from acute nutritional disturbances he finds a lessening of tolerance. Modigliani and Benini[92] have made similar tests to determine whether the casein of cow's milk ever passes the intestinal wall unchanged. They obtained positive results in the case of infants, both new-born and older infants with gastro-intestinal disturbances who were fed on cow's milk.

The question of the presence of casein and paracasein in the stools of infants has been studied still further by Poulsen,[93] Uffenheimer[95] and Liwschiz.[96]

INFLUENCE OF BACTERIA ON NUTRITIONAL DISTURBANCES

97. Day and Gerstley: Am. Jour. Dis. Child., 1915, ix, 233.
98. Young: Arch. Pediat., 1914, xxxi, 753.
99. Breuning: Jahrb. f. Kinderh., 1914, lxxx, 98.
100. Metchnikoff: Ann. de l'Inst. Pasteur, 1914, xxviii, 89.
101. Bertrand: Ann. de l'Inst. Pasteur, 1914, xxviii, 121.
102. Bertelot: Compt. rend. hebd. de l'Acad. d. sc., 1913, clvi, 1567.
103. Blühdorn: Monatschr. f. Kinderh., 1914, xiii, 37.
104. Schild: Monatschr. f. Kinderh., 1914, xiii, 51.
105. Schelble: Jahrb. f. Kinderh., 1914, lxxix, 507.
106. Armstrong: Jour. Am. Med. Assn., 1914, lxiii, 200.
107. Bobillier: Thèse de Paris, 1913, No. 327; Ztschr. f. Kinderh. (ref.), 1914, viii, 191.
108. De Buys: Jour. Am. Med. Assn., 1914, lxiii, 1806.
109. Archibald: Jour. Trop. Med., 1914, xvii, 161.
110. Morse and Talbot: Boston Med. and Surg. Journal, 1915, clxxii, 171.
111. Peyri-Rocamora: rev., Arch. Pediat., 1915, xxxii, 159.
112. Logan: Jour. Path. and Bacteriol., 1914, xviii, 527.
113. Editorial: Jour. Am. Med. Assn., 1914, lxiii, 952.
114. Basten: Ztschr. f. Hyg. u. Infectionskrankh., 1914, lxxvii, 282.

That bacteria may cause gastro-enteritis in infants seems now to be a well-established fact; especially is this true of the type of case showing symptoms resembling dysentery. Still unsettled, however, is the question of the relative importance of bacteria in all cases of diarrhea. Morse and Talbot[110] believe that when large numbers of bacteria of the dysentery group or streptococci are found in the stools of cases showing marked disturbance of the intestinal tract, they are in most instances the cause. The presence of a few of these organisms in the stools in diarrhea does not, however, prove that they are the cause, because they may be present in small numbers in the stools of normal infants.

Day and Gerstley[97] have attempted to reconcile the conflicting theories of the cause of infantile diarrhea by showing that different types of cases have different causes. Twenty-two cases of severe diarrhea observed by them are classified into: food disturbances, of which there were two; cases of infectious diarrhea, of which, also, there were but two; and parenteral infections, eighteen cases. The cases of infectious diarrhea were caused by the gas bacillus.

Young[98] reviews the research work of the Boston Floating Hospital during the last few years on the subject of infectious diarrhea in infants. Though clinically the cases show the same picture, they may be divided according to the cause into cases caused by the dysentery bacillus, and those caused by the gas bacillus. The treatment varies according to the cause. In cases caused by the dysentery bacillus, after sterile water for twenty-four hours a 5 per cent. lactose solution is given until the temperature is normal and blood has disappeared from the stools. In extreme cases 2.5 per cent. dextrose

solution is given subcutaneously. Cases caused by the gas bacillus, on the other hand, require a food containing little sugar and a high protein content. The observations of different years show a seasonal variation both in the type of organism and in the degrees of virulence of the same organism.

Breuning[99] examined 117 cases of diarrheal disease in infancy and early childhood for the presence of "paratyphus B." as the cause. In eleven cases the serum was found to agglutinate this bacillus. Three patients were under 1 year. One baby of 6 months developed a metastatic abscess in the shoulder which showed a pure culture of "paratyphus B." bacilli. Breuning advises that more frequent bacteriological examinations should be made in case of acute gastro-enteritis in infants.

Several French observers believe that the *Bacillus proteus vulgaris*, especially in conjunction with other bacteria, may frequently cause infantile diarrhea. Metchnikoff[100] has come to this conclusion, after a series of bacteriologic examinations of stools of infants showing intestinal disturbances. This etiologic significance is present especially when the *B. proteus vulgaris* is associated with another of the intestinal bacteria. The *B. proteus* was also found in normal infants who acted, according to Metchnikoff, as carriers.

Among fifty-five cases of diarrhea examined by Bertrand[101] in the summer of 1912, the *B. proteus vulgaris* was found in all; among twenty-four normal children only twice.

Bertelot[102] agrees with the theory of Metchnikoff.

Several authors have described cases of bacillary dysentery. Blühdorn[103] during 1912 and 1913 saw twenty-four cases in infants and young children. The clinical symptoms were frequently the same as those of disturbances caused by food; severe cases especially, showed a marked resemblance to cases of alimentary intoxication. The cases of dysentery were, however, markedly contagious; sugar was absent from the urine, and breast-fed children were frequently attacked. In different epidemics the following types of cases were observed: simple intestinal catarrh; cases associated with a grip infection; cases without fever, but showing mucus and sometimes blood in the stools; and very severe cases, occurring usually in atrophic children with symptoms either of decomposition or of intoxication. In the treatment, after emptying the gastro-intestinal tract, sterile water is given for a short period, which should not be continued too long in cases debilitated by a long continued cereal water diet. Whey has been used for several years, with good results. Blühdorn advises an examination for the bacilli in all cases suspicious of dysentery. In intoxication the disease should be suspected if the condition does not yield when all food is withdrawn.

Schild[104] made the bacteriologic and serologic examinations, determining the diagnosis in the cases reported by Blühdorn. The bacilli found belonged culturally to the group of pseudo-dysentery bacilli. Some of these strains were agglutinated by well-known races; those isolated from cases in one epidemic, however, could not be identified with any known race of pseudo-dysentery bacilli either by complement fixation or agglutination. Complement fixation was found to be a very sensitive test for the identification of different strains of bacteria.

Schelble[105] reports sixteen cases of acute nutritional disturbances occurring simultaneously in the form of an epidemic among the twenty infants in an institution. Parenteral infection in these cases could be ruled out as a causative factor; the author agrees, however, with Meyer that this is the most frequent factor in nutritional disturbances of infants in institutions. All sixteen infants affected were taking the same milk; the four infants spared did not receive this milk. The stools of the infants affected showed no typhus, paratyphus or dysentery bacilli, and agglutination tests were negative. The author was undecided as to the cause of this epidemic.

The influence of flies in causing infantile diarrhea through the carrying of infection has been the subject of much popular propaganda; the experimental studies in proof of this theory are, however, very few. That carried out by the Bureau of Public Health and Hygiene of the New York Association for the Improvement of the Poor and reported by Armstrong[106] is therefore especially interesting. In the Italian district of the Bronx two areas were selected; in each there were 311 families with 1,725 individuals in one area and 1,744 in the other. The general conditions in the two areas were similar, and there were about the same number of breast-fed infants in each. In one area all known measures for protection against flies were taken; doors and windows were screened, manure was treated with iron sulphate, fly traps were placed, oral and written instructions were given, supplemented by moving picture films. Records of morbidity in the two areas for the period between July 21 and September 13 are interesting. The cases of diarrhea in children under 5 years were twenty in the area protected against flies as compared with fifty-seven in the unprotected area; the total number of days of sickness among these children from this cause were 273 in the protected area and 984 in the unprotected. Armstrong emphasizes the necessity of continuing the investigation on a larger scale and for a longer time.

Amebic dysentery, according to Bobillier[107] is very rare even in the tropics in infants under 2 years, but has been observed in France. Emetin works well in small doses. DeBuys[108] reports eight cases in

young children; the two youngest were 4 years old. The treatment was emetin.

Archibald[109] saw two patients aged 2½ years and 8 months in the same family. For a 2-year-old child he advises emetin 0.01 gm.

Bacteriology of the Normal Stools

Morse and Talbot[110] have summarized the literature in regard to the bacteriology of the gastro-intestinal tract in infancy. Peyri-Rocamora,[111] Logan,[112, 113] and Basten[114] have studied this subject.

INFLUENCE OF PARENTERAL INFECTIONS ON NUTRITIONAL DISTURBANCES

115. Langstein: Jahresb. f. ärztl. Fortbild., 1914, v, 57.
116. Stolte: Jahrb. f. Kinderh., 1914, lxxx, 213.
117. Triboulet: Med. Infant., 1914, xxii, 73.
118. Grünfelder: Ztschr. f. exper. Path. u. Therap., 1914, xvi, 141.
119. Langstein: Ztschr. f. Kinderh. (ref.), 1914, viii, 54.
120. Czerny: Ztschr. f. Kinderh. (ref.), 1914, viii, 144.
121. Kowitz: München. med. Wchnschr., 1914, lxi, 1321.

The work of Meyer on "hospitalism" was reviewed last year. Langstein[115] refers to this work as proving that the bad influence of hospitals on infants does not depend solely on infections of the intestinal tract or on insufficient care; but depends especially on infections of the respiratory passages ("grip"). These infections predispose greatly to nutritional disturbances, and are so prevalent in infants' institutions that hardly a child remaining for some time in the institution is spared. He writes that nutritional disturbances when occurring together with "grip" should be treated not by taking away the food, but by feeding with albumin milk.

These views of Langstein and Meyer with regard to the cause of "hospitalism" in infants are questioned by Stolte.[116] He believes that more important than infections, in causing the poor results with infants in hospitals, is the lack of personal and individual care. An experiment was made in the children's clinic at Berlin to imitate the care usual in a private home; each infant was put into the special care of one nurse, who was instructed to give wide play to the infants' likes and dislikes as to food. The results were excellent; there was gain in weight, as well as improvement in general condition, color and activity. He concludes that it is possible in a hospital by using this method to achieve as good results as those obtained in private homes.

Triboulet[117] writes of the predisposition to nutritional disturbance associated with certain infections. In uncomplicated pneumonia he believes that the intestine is not necessarily affected; but in severe

pneumonia he has found a regular connection with diarrhea, as also in cases of pneumonia complicated by purulent pleuritis, otitis and peritonitis.

Grünfelder[118] has studied experimentally in dogs the effect of acute infections on the secretion of gastric juice. The formation of Pawlow's "blind stomach" was accomplished in two dogs; fever and infections were induced by causing localized abscesses or by insufflating infectious material into the nostrils. The amount of gastric juice secreted during the infections was found to be greatly reduced; it was more slowly secreted, and showed a diminution in hydrochloric acid. Grünfelder believes that these findings are parallel to the nutritional disturbances occurring in infants in the course of acute infections.

Pyelocystitis is referred to by Langstein[119] as being very frequently associated with anorexia, which may be so marked that feeding by gavage must be resorted to.

The fact that nutritional disturbances likewise predispose to certain infections is referred to by Czerny,[120] especially with regard to paravertebral pneumonia. Kowitz[120] believes that acute alimentary disturbances predispose greatly to pyelocystitis in infants.

RELATION OF HEAT AND OTHER FACTORS TO INFANT MORBIDITY AND MORTALITY FROM NUTRITIONAL DISTURBANCES IN SUMMER

122. Schreiber and Dorlencourt: Arch. de méd. d'enf., 1914, xvii, 1.
123. Breyer: Ztschr. f. Kinderh. (ref.), 1914, viii, 158.
124. Prinzing: Wien. klin. Rundschau, 1913, xxvii, 678.
125. Japha: Ztschr. f. Kinderh., 1913, vii, 518.
126. Heiman: Arch. Pediat., 1914, xxxi, 466; Am. Jour. Dis. Child., 1914, viii, 138.
128. Helmholtz: Jour. Am. Med. Assn., 1914, lxiii, 1371.
129. Nicolaysen: Monatschr. f. Kinderh. (ref.), 1914, xiv, 10.
130. Rohmer: Ztschr. f. Säuglingfürs., 1914, vii, 329, 385.
131. Epstein: Beth. z. med. Klin., 1913, No. 9.

For the last few years many studies have been made, especially by German authors, on the subject of the direct action of hot weather in causing nutritional disturbances in summer in infants.

Schreiber and Dorlencourt[122] have observed in young dogs exposed to very high temperatures the same symptoms as in infants with heat prostration.

Many German authors have shown in the last year that housing conditions which favor a high indoor temperature predispose to infantile diarrhea. Breyer[123] and Prinzing[124] again bring forward evidence in proof of this theory. Japha,[125] from results in work at infant consultations, concludes that pure milk cannot protect infants in summer from the dangerous effects of high temperatures, and that

housing improvements and instruction in general hygiene are necessary for this purpose. Heiman[126, 127] has made the experiment of cooling artificially in summer a ward used for infants with nutritional disturbances. The temperature of the room was during the forty-two days of the experiment from 5 to 11 degrees below the outside temperature. Thirteen patients were treated; none of these were premature or moribund infants, as in such cases the treatment seems not to be indicated. One infant died, twelve recovered. Control observations could not be made. Heiman gained the impression that the infants so treated were more comfortable than infants in the other wards.

Helmholtz[128] has made an interesting study of the relation of high indoor temperatures to high body temperature of infants, and to the mortality and morbidity of infants in summer. Maximum and minimum temperatures were recorded in the homes of the forty-six infants studied; a daily call by a nurse was made, the condition of the infant was noted and its temperature taken. In the thirty-nine attacks of dyspepsia observed in twenty-nine infants, there seemed no definite relation between high room temperature and gastro-intestinal disturbance. The average room temperature of the day before and of the day of the onset of the dyspepsia was 89 F. Very high maximum indoor temperatures were observed; in ten cases temperatures over 105 were recorded. The indoor temperatures exceeded the outdoor frequently by marked amounts; once by 40 degrees and five times by 30 degrees. There was, however, no constant relationship between fever in the infant and a high room temperature; as in 80 per cent. of the cases the infants showed in spite of a high temperature in the house a normal range of body temperature. Among the six cases which terminated fatally, only one or two could be ascribed to the direct effect of heat. Helmholtz calls attention to the fact that the clothing of infants varies greatly, and that clothing may act very intensely in producing fatal heat retention, this having been shown recently for experimental animals in the Sprague Institute Laboratory by Drs. McClure and Sauer. He concludes that the study would indicate that an improper adjustment of the individual to the surrounding temperature, rather than the height of the temperature, increases the mortality from gastro-intestinal diseases in summer; and that the aim should be to educate mothers to keep their infants cool in summer.

Nicolaysen[129] has analyzed the statistics of the deaths of infants from diarrhea in Christiania. He found that several such deaths frequently occurred in the same house in the same year. Of six houses in which especially many cases occurred, only one showed

conditions which might lead to overheating; the remaining five showed good ventilation. He concludes that these findings speak for an infectious cause of infant diarrhea, and believes that flies are very important in spreading the disease.

The work of Armstrong[109] with regard to the relation between flies and infantile diarrhea has been mentioned.

Rohmer[130] has made an interesting investigation of the relative importance of different forms of feeding and of housing conditions in causing infant diarrhea in summer. In a number of streets of the older section of Cologne, where the infant mortality was very high, he visited regularly all the infants born, and noted the conditions. Breast-feeding he found to be fairly prevalent; 40 per cent. were breast-fed for nine months. Of children breast-fed in the second half year, 80 per cent. were healthy; of infants not breast-fed or insufficiently breast-fed, 18 per cent. only were healthy. As the summer was a cool one the relation of the temperature of the house to infant diarrhea could not be studied. Rohmer believes that the house itself is not most important, but rather the fact of how the house is used and how the baby is cared for.

Epstein[131] considers as still unsettled the question as to the cause of the action of summer heat; he believes that a high summer temperature is only one factor together with a number of other causes.

NOTES ON NUTRITIONAL DISTURBANCES

132. Czerny: Jahrb. f. Kinderh., 1914, lxxx, 601.
133. Schloss: Arch. Pediat., 1914, xxxi, 924.
134. Knox and Tracy: Am. Jour. Dis. Child., 1914, vii, 409.
135. Holt: Arch. Pediat., 1914, xxxi, 455.
136. Holt, Courtney and Fales: Am. Jour. Dis. Child., 1915, ix, 213.
137. Finkelstein: Ztschr. f. ärtzl., Fortbild., 1914, xi, 101.
138. Bernard: Dissertation, Leipzig, 1913; Ztschr. f. Kinderh. (ref.), 1914, viii, 127.
139. Hess: Jour. Am. Med. Assn., 1914, lxii, 452.

Czerny[132] has studied the heart findings in infants with acute nutritional disturbances. A phenomenon which has been much discussed is the fact that before death in certain cases only one heart tone is heard. Czerny has always maintained that this tone is the first.

Among 195 cases of infants with nutritional disturbances varying from the mild to the severe type, Schloss[133] found twenty-seven cases with sugar in the urine. This sugar was usually galactose or dextrose. A hyperglycemia accompanied the melituria in most of the cases.

Knox and Tracy[134] have determined the excretion of phosphates in the urine of infants on various forms of feeding, and in the course of nutritional disturbances. They conclude that deductions as to the

nature and severity of such disturbances cannot be drawn merely from the amount of urinary phosphorus present.

The chemical analyses of the stools of infants with disturbances of nutrition, as well as those of normal infants, have been made by Holt,[135] and by Holt, Courtney and Fales.[136] Loose stools contained from about 100 to 200 c.c. of water per day, very loose stools over 200 c.c. The fat in very loose stools was about two and a half times that in normal stools; 64 per cent. of the fat was neutral fat in loose stools as compared with 39 per cent. in normal stools. About two and a half times as much protein was lost daily in loose stools as in normal. Of the salt elements the chlorin, potassium and sodium were much higher in proportion in the former than in the latter; 84 per cent of the total intake of ash was lost in loose stools, as compared with 40 per cent. in normal stools. The latter striking condition is commented on by the authors as giving suggestions for treatment.

Alimentary Intoxication

Finkelstein,[137] in a summary of his teachings with regard to the various disturbances of nutrition in infancy, states his belief that both decomposition and intoxication are due to disturbances in the metabolism of water. In the treatment the attempt must be made to stop the loss of water, and to favor a new storing up of water.

Bernard[138] found that an increase in the refractometric value of the blood is the rule in intoxication. He believes that this is due to an excretion of water in excess of the excretion of salt.

In severe gastro-intestinal disturbances of infants, especially in intoxication, Hess[139] advises the drop method of giving fluid by mouth, as aiding the administration of the large amount of water necessary in this condition. The apparatus used is similar to that in common use in the instillation of liquid into the rectum, except that a rubber nipple with a small hole is substituted for the hard rubber rectal tip. The flow from the nipple can be so adjusted that the child receives from 25 to 30 drops per minute. The infant is encouraged to suck at the nipple the greater part of the day. Water or hypotonic salt solution may be given.

LACTIC ACID BACILLI

140. Berry: Arch. Pediat., 1914, xxxi, 525.
141. Bendick: Jour. Am. Med. Assn., 1915, lxiv, 809.
142. Miscellany: Jour. Am. Med. Assn., 1914, lxii, 1835.

Berry[140] has used cultures of lactic acid bacilli in twenty-four cases of acute intestinal indigestion characterized by slight fever, vomiting and liquid stools. Some of the patients were treated simply by the administration of the bacilli; others were treated by change

in diet, and medicines in addition. He concludes that the best results were obtained when the tablets were used together with a proper change in the diet and an initial dose of castor oil. The bacilli were administered in the commercial tablets, one tablet four times daily, or in severe cases one tablet every three hours. The classification of such cases as those treated as cases of putrefactive fermentation is at variance with most theories in regard to the etiology of this type of dyspepsia.

The commercial preparations of the *B. bulgaricus* have been studied by Bendick.[141] He has found many specimens sterile on culture; the majority show but a small fraction of the number of living organisms represented by the manufacturer. A broth culture is the most active form of preparation. He recommends that preparations should be marked with the date of manufacture, and should always be kept in the ice-box.

A bulletin of the Department of Agriculture[142] discusses preparations of lactic acid bacilli. The most successful commercial preparations are the liquid forms, but dry forms are more convenient for transportation. A method devised by the Department of Agriculture consists in freezing cultures and then drying them over sulphuric acid.

GASTRIC DIGESTION OF INFANTS

144. Hahn: Am. Jour. Dis. Child., 1914, vii, 305.
145. Schackwitz: Monatschr. f. Kinderh., 1914, xiii, 73.
146. Davidsohn: Monatschr. f. Kinderh., 1914, xiii, 182.
147. Aron: Jahrb. f. Kinderh., 1914, lxxix, 288.
148. Huenekens: Ztschr. f. Kinderh., 1914, xi, 297.
149. Ladd: Boston Med. and Surg. Jour., 1914, clxx, 518.

Many publications this year have been on the subject of the concentration of hydrochloric acid in the infant's stomach, and of the possibility of peptic digestion.

Hahn[144] reviews some of the previous work on this subject. Pepsin is activated in the stomach by a certain concentration of hydrochloric acid. The concentration of acid depends on the concentration of dissociated hydrogen ions; this cannot be determined accurately by titration. The methods of determination used by Michaelis and Davidsohn, the indicator and electrometric methods, are reliable. The concentration of hydrogen ions is expressed by the symbol (H), in gram ions. It has become customary to abbreviate the small decimal fractions used by means of a negative coefficient of 10; for instance $10^{-1} = 0.1$; $10^{-2} = 0.01$; $10^{-5} = 0.00001$, etc. The optimum (H) for pepsin digestion is 1.6×10^{-2}, while at (H) 1.0×10^{-5} pepsin is inert. The latter is, however, the optimum reaction for rennet and gastric lipase. Hahn has found the concentration of 1.0×10^{-5} quite constantly in the stomach contents of artificially fed infants at the height of

digestion. Determinations were made on ninety-four specimens from thirty-seven infants on artificial feeding (one-third cream-milk and two-thirds milk). The stomach contents for examination were removed by catheter as completely as possible, at the height of digestion.

Schackwitz[145] considers that the examinations previously made by Davidsohn and others have been too few to prove that the concentration of hydrochloric acid is insufficient for peptic digestion in the infant's stomach. He agrees that the physicochemical method of determination is the only accurate method; and has used this method in 137 examinations of the stomach contents of sixty infants. He finds the concentration to vary between very wide limits, so that he cannot agree with Davidsohn that the low concentration $(H) = 1.0 \times 10^{-5}$ is constant in infants.

Davidsohn[146] responds by calling attention to the work of Hahn,[144] confirming his own earlier findings. He explains the difference in results by the fact that while he and Hahn regularly removed for examination all of the stomach contents obtainable, Schackwitz removed only 1 or 2 c.c. As the degree of acidity may differ widely in different regions of the stomach, this method may lead to error.

Aron[147] has suggested that in explaining the low concentration of acid found in the infant's stomach we must consider whether there is an insufficient secretion by the stomach, or whether the acid is not combined with the milk. In experiments to test the power of cow's milk and of human milk to bind acid, he has found that cow's milk binds much more acid than human milk. He draws attention to the fact that Hess found free hydrochloric acid present in the stomach of a new-born child before any food was taken. Aron believes that it is chiefly the binding of acid by milk which determines the low acidity usually present in the stomach contents of the infant.

Huenekens[148] has studied the acidity of the gastric contents in infants and young children when meat is given. Examinations were made on five children, aged respectively 9½, 13, 17 and 20 months and 5 years. Determinations of the hydrogen ion concentration according to Davidsohn were made. In each case one determination was made after a feeding with milk, and one after a meat and vegetable feeding. After the milk feeding a low degree of acidity was found for two reasons: milk has little stimulating power on acid secretion, and it binds acid to a marked degree. After the feeding with meat the acidity in the stomach contents of the infants 9½ and 13 months old was slight, so that no peptic digestion could occur; in the three older children, however, there was a marked increase in the concentration of acid. Huenekens concludes that without special

reason meat should not be given to children before the second half of the second year.

Ladd[149] has continued his studies of the motility of the infant's stomach as shown by Roentgen ray. The administration of a large amount of alkaline fluid hastens the emptying of the stomach; this was observed when from 25 to 50 per cent. of the whole feeding was lime water, or when 0.84 per cent. of sodium bicarbonate to 200 gm. of milk mixture was given.

PYLORIC OBSTRUCTION

150. Holt: Jour. Am. Med. Assn., 1914, lxii, 2014.
151. Reuben: Arch. Pediat., 1914, xxxi, 809.
152. Langmead: Proc. Roy. Soc. Med., 1914, vii, 93.
153. Cautley: Proc. Roy. Soc. Med., 1914, vii, 92.
154. Hess: Am. Jour. Dis. Child., 1914, vii, 184.
155. Hess: Ergebn. d. inn. Med. u. Kinderh., 1914, xiii, 530.
156. Hess: Am. Jour. Dis. Child., 1914, vii, 428.
157. Peiser: Monatschr. f. Kinderh., 1914, xiii, 121.
158. Reuben: Arch. Pediat., 1914, xxxi, 782.
159. Ruhräh: Am. Jour. Med. Sc., 1914, cxlvii, 474.
160. Richter: Jour. Am. Med. Assn., 1914, lxii, 353.
161. La Fétra: Arch. Pediat., 1914, xxxi, 761.
162. Downes: Jour. Am. Med. Assn., 1914, lxii, 2019.
163. McKechnie: Canad. Med. Assn. Jour., 1913, iii, 566.
164. Lewis and Grulee: Jour. Am. Med. Assn., 1915, lxiv, 410.
165. Scudder: Ann. Surg., 1914, lix, 239.
166. Lillienthal: New York Med. Jour., 1914, xcix, 723.
167. Hougardy: Ztschr. f. Kinderh. (ref.), 1914, viii, 511.
168. Liefman: Monatschr. f. Kinderh., 1914, xii, 714.
169. Oberwarth: Ztschr. f. Kinderh. (ref.), 1914, viii, 18.
170. Lichtenstein: Dissertation, Berlin, 1913: Ztschr. f. Kinderh. (ref.), 1914, viii, 232.
171. Knöpfelmacher: Ztschr. f. Kinderh. (ref.), 1914, viii, 77.

Diagnosis of Hypertrophic Stenosis and Pylorospasm

The difficulty of the differentiation of cases of pyloric obstruction into cases of pylorospasm or of organic stenosis is commented on by most of those writing this year on this puzzling subject.

Holt[150] has proposed to substitute for the preceding classification a division of these cases into mild and severe. He points out the fact that there has always been great difference of opinion as to the symptoms which may be considered diagnostic of pylorospasm and those diagnostic of organic stenosis. The suggestion that a palpable tumor is a sign diagnostic of stenosis has not been accepted; nor has the theory that recovery without operation necessitates the diagnosis of pylorospasm. Holt believes that a definite persistent spasm of the pylorus without stenosis has yet to be proved, though a temporary spasm may occur. The two elements of spasm and hypertrophy are in his opinion present in each case, the essential difference in the cases

being one of degree only. The term "pylorospasm" should, therefore, be dropped, as it has led to confusion, especially in regard to indications for operation. He analyzes the symptoms of pyloric obstruction on the basis of fifty-seven cases observed by him. The great majority of these infants (fifty-two) were breast-fed; forty-nine were males.

The suddenness of the onset was noted in thirty cases, in many almost the hour of the beginning of the trouble being fixed. This fact he ascribes especially to the important part played by spasm. Vomiting began in four-fifths of his cases in the third, fourth and fifth weeks. The tumor he found to be more easily palpable during or immediately after vomiting; it persisted in some cases for weeks and even months during convalescence. The general mortality in his cases was 55 per cent.; of twenty-eight patients operated on fourteen died; of twenty-nine on medical treatment seventeen died. The indications for operative treatment he believes depend on the degree of severity of the obstruction; this is shown by the continuance of vomiting and loss in weight, and by the gastric retention, measured by aspiration of the stomach three hours after a meal. Roentgenoscopy he has found to be misleading. In the medical treatment he advises lavage twice daily, and careful feeding with a breast-milk not rich in fat; from 1 to 3 ounces of milk every three to four hours are usually given. After operation hypodermoclysis of normal saline solution with 4 per cent. dextrose is useful. Feeding with breast-milk should be begun after four hours; at first 2 drams every two hours are given; later the amount is gradually increased.

Holt's suggestion that the cases should be classified as mild or severe is considered very useful by Reuben,[151] as he believes that the treatment depends on the severity of the case. In his opinion, however, two distinct types of pyloric obstruction occur, the spasmodic and hypertrophic, although their differentiation is not important for treatment. In proof of this fact he states that a few of the cases which during life have shown all the symptoms of pyloric stenosis, have presented at necropsy a perfectly normal pylorus. Speaking for spasm are a neuropathic history, presence of bile in the vomitus, intermittence of the symptoms, sudden and spontaneous cure, absence of palpable tumor, or, if a tumor is present, its variation in size and shape. Reuben uses Hess' duodenal tube as an aid to diagnosis; he has found in spasm a resistance at the same point, which is felt to relax suddenly. Failure to pass the catheter after repeated attempts means a marked stenosis; a mild degree of stenosis cannot be distinguished from spasm. He considers that operation is contraindicated when the duodenal catheter can be passed.

In the discussion of a case reported by Langmead,[152] where the presence of vomiting, visible peristalsis and a palpable pylorus were sufficient in his opinion to show that a true stenosis existed, Cautley[153] expressed his belief that these symptoms were not definite evidence that a hypertrophic stenosis was present. Hess[154] remarks on the great difficulty found by all writers on the subject in diagnosis of pylorospasm from organic stenosis or simple vomiting. He uses as a test the passage of a No. 15 (F.) catheter into the duodenum; and in all cases in which this is possible, assumes that organic stenosis is so slight that it can be disregarded from a clinical point of view. Elsewhere[155] he remarks that failure to pass the catheter after repeated attempts does not absolutely exclude spasm; in two or three cases of spasm he has been unable to enter the duodenum. As an aid to passing the catheter into the duodenum in cases of spasm he advises[155] the giving of a little water through the tube into the stomach, after which the pylorus may frequently be passed. He has reported twenty cases of pylorospasm. In not one of these was a palpable tumor present, and he believes that tumor has never been noticed where hypertrophy was not found. He calls attention to the fact that pharyngospasm and cardiospasm are frequently associated with spasm of the pylorus. Visible peristalsis, he believes, is not necessarily a sign of organic stenosis, or even of pylorospasm; he has noticed it in cases in which neither was present.

Peiser,[157] in his use of Hess' duodenal catheter, has found difficulty in proving that the duodenum has been entered, and considers that the successful passage of the tube does not always contraindicate operation. In disagreement with Hess' statement that palpable tumor of the pylorus is associated only with hypertrophic stenosis, Reuben[158] reports a case which he diagnosed as pylorospasm, in which the duodenal catheter was passed and recovery later occurred though a palpable tumor was noticed; and Liefman,[168] among forty-six cases of pyloric spasm, noted a tumor in six cases.

Ruhräh[159] believes that a change in the size of the tumor under the palpating finger is a sign that the obstruction is spasmodic, or at any rate that spasm is the most important element. He advises operation as early as possible in cases of hypertrophic stenosis.

One can but agree with Holt that there is as yet no agreement in regard to the essential diagnostic signs and symptoms of spasm and hypertrophic stenosis.

Hypertrophic Stenosis and Operative Treatment.

Richter,[160] however, believes that the cases can be distinguished after a long enough period of observation, and that borderline cases do not occur. Of the twenty-two cases in which the author operated

(eleven having been previously reported) nineteen were organic stenosis, three pyloric spasm. Among his cases also, boys were in the majority; all were first-born except one. The onset of vomiting was in the second or third week and was sudden. He considers the Roentgen-ray examination misleading, as the entrance of bismuth into the intestine does not speak against hypertrophic stenosis. The mortality of his cases was very low, being 13 per cent. The eighteen children who recovered developed normally.

La Fétra,[161] too, states that he has found the Roentgen ray misleading and unessential in the diagnosis of pyloric stenosis.

Downes[162] considers that operation is indicated whenever a diagnosis of hypertrophic stenosis has been made; but operation should not be performed in cases of pyloric spasm.

According to MacKechnie,[163] the two conditions of spasm and organic stenosis are distinct, but an absolute diagnosis can not always be made by palpation; therefore he advises an operation in cases with marked decrease in weight. He reports six cases treated surgically.

Lewis and Grulee[164] report the pathologic findings at the pylorus in an infant who died of pneumonia eight months after a successful operation. An enlargement at the pylorus was found of approximately the same size and consistency as of that seen at operation, indicating that no change had occurred in the tumor. They believe that gastro-enterostomy is well borne by infants if performed rapidly and without trauma. Among five patients operated on there were no deaths.

Scudder[165] reports seventeen operative cases, nine of which had been reported before. There were three deaths, making a mortality of 17 per cent. A table shows the normal development of those who recovered, one having been observed for eight years. Roentgen examination in these children showed that the food continued to pass through the operative opening.

Lillienthal[166] and Hougardy[167] have reported cases of stenosis with recovery after operation.

Pylorospasm

Hess[154] has reported twenty cases of spasm. He has described[156] a duodenal catheter furnished near its tip with a small collapsible balloon, which may be inflated with air after the introduction of the catheter into the duodenum, thus effecting the retention in place of the catheter for as long as twenty-four hours. He has used this catheter in a few mild cases of spasm for duodenal feeding; he has not yet made enough experiments with pronounced cases of pylorospasm to be able to draw conclusions.

Liefman[168] has studied the symptoms and history of forty-six cases of pyloric spasm and six cases of habitual vomiting occurring during three years in the Dresden Saüglingsheim. The mortality was 10.8 per cent. Forty per cent. of the cases were among girls; only fifteen patients were breast-fed; twenty-eight were first-born children. The onset was from the first to the sixth week, twelve cases beginning in the third week. That a pyloric tumor was present in six cases has been mentioned. In the latter history neuropathic symptoms were noted only in one-half the cases. The general development was normal.

Oberwarth[169] reports twenty cases of spasm; all the patients recovered on medical treatment. He considers important the regular rectal instillation of 200 gm. of breast-milk daily.

Ruhräh[159] in cases of pylorospasm advises stomach washing several times daily, using 0.8 per cent. sodium bicarbonate solution if much mucus is present. He considers atropin the best medical treatment.

Lichtenstein[170] reports fifteen cases of pylorospasm in infants. Knöpfelmacher[171] reports a case of pylorospasm favorably influenced by papaverin.

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